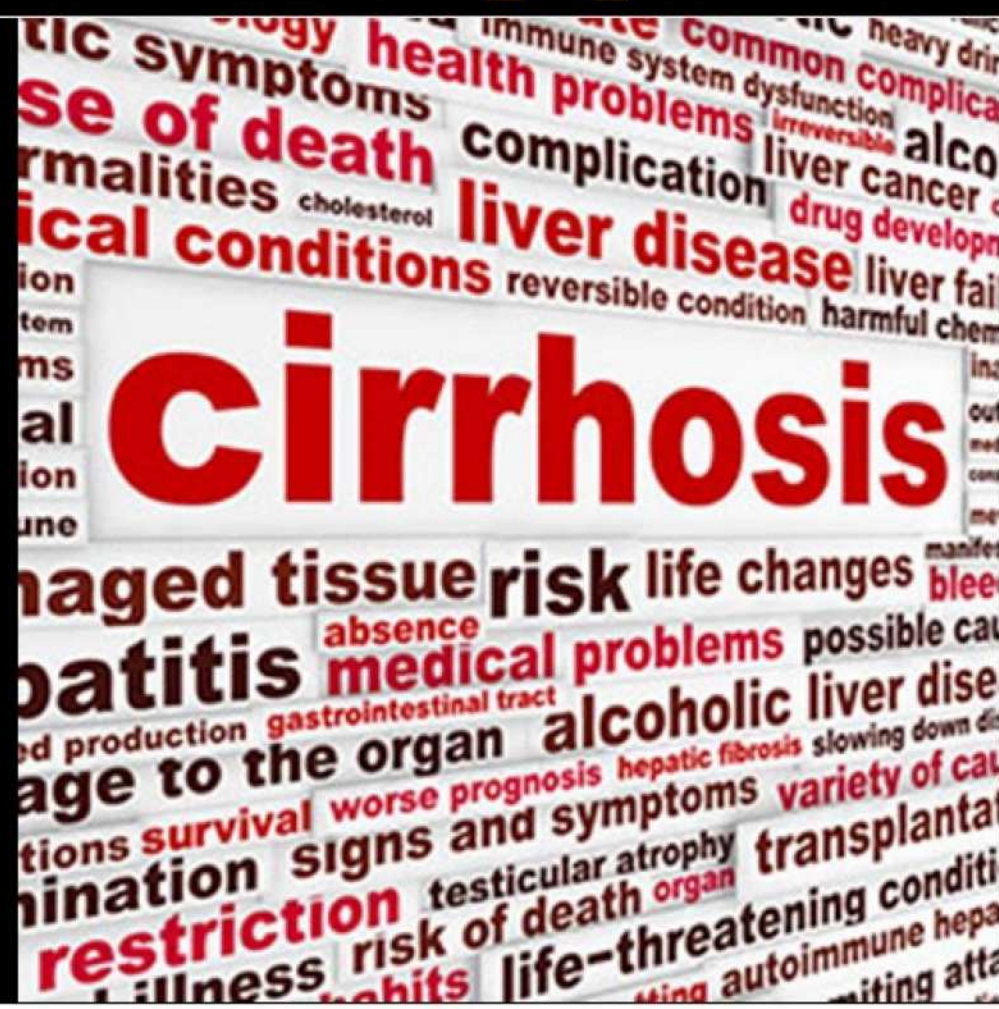


Liver Cirrhosis [1]



VISIT...

LANZAROTE
Caliente.COM

تحذير هام

هذا الفيديو و جميع الرسوم التوضيحية المتاحة فى هذا الموقع هم تحت حماية قانون الملكية الفكرية

هذا الفيديو و جميع الرسوم التوضيحية المتاحة فى هذا الموقع هم تحت حماية قانون الملكية الفكرية و لا يحق لأحد إستخدامه او تحميله أو إستنساخه أو إعادة طبعه أو طبع أجزاء منه بدون إذن مسبق من الدكتور طارق عبد الحميد (أو من ينوب عنه قانونياً). و من يتعدى على الملكية الفكرية المذكورة فسوف يتعرض للمسائلة القضائية عن جريمة إنتهاك حقوق الملكية الفكرية. و إستخدام الفيديوهات و المادة العلمية المذكورة مكفول فقط لمستخدم واحد فقط و لأخذ تصريح إستخدام لأكثر من شخص برجاء الإتصال بالدكتور طارق عبد الحميد

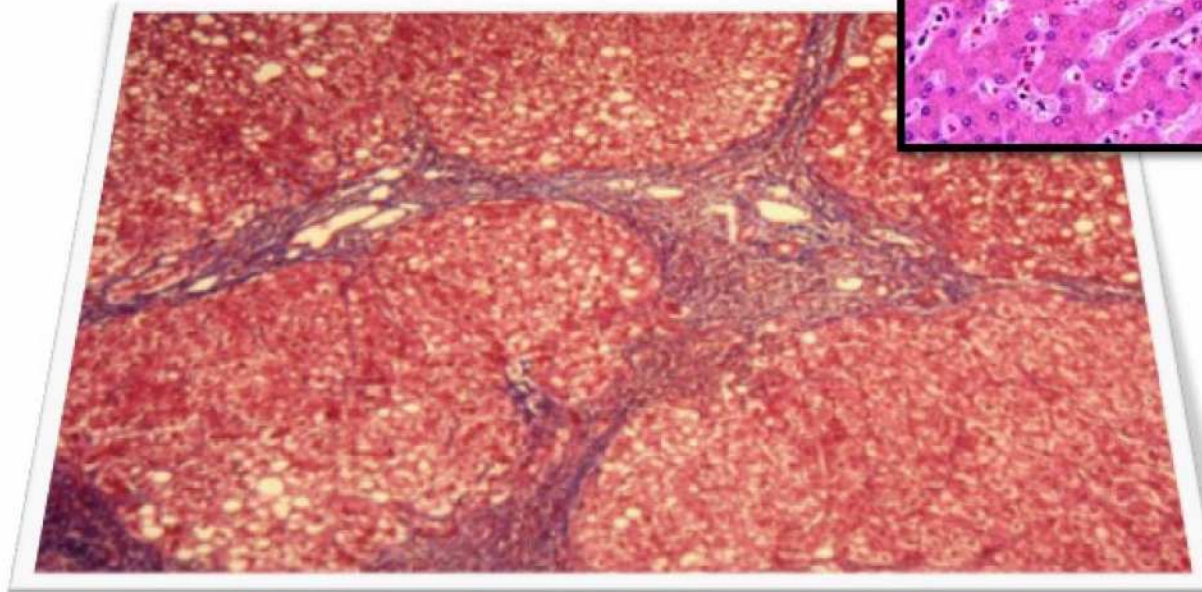
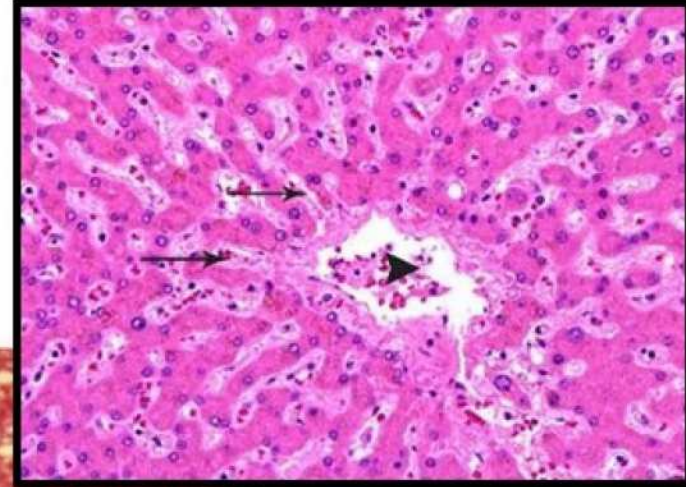


Definition

A Chronic diffuse hepatic condition characterized by:

- ① Degeneration
- ② Regeneration
- ③ Fibrosis
- ④ Loss of architecture

Normal Liver

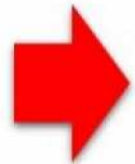
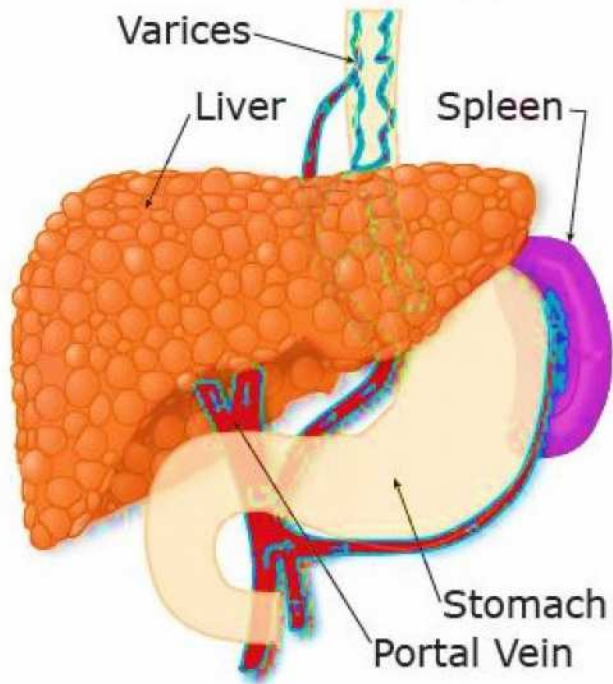


Cirrhotic Liver

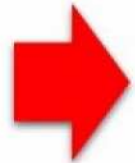
Aetiology

- 1- Alcoholic liver disease**
- 2- Post-Viral hepatitis (HBV, HCV)**
- 3- Biliary Cirrhosis**
- 4- Hemochromatosis**
- 5- Wilson disease**
- 6- Alpha 1 Anti-trypsin defficiency**
- 7- Cryptogenic or Idiopathic cirrhosis.**

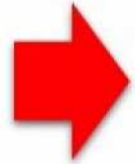
Clinical Picture



Liver cell failure

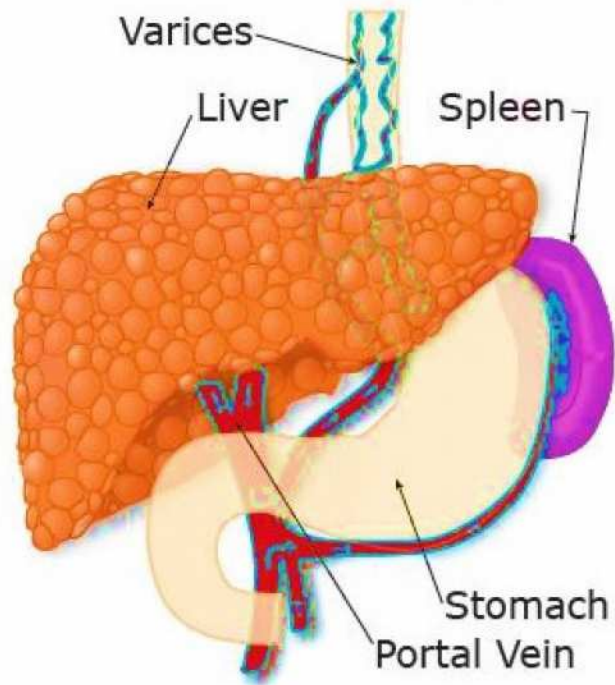


**Portal
hypertension**

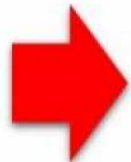


Hepatoma

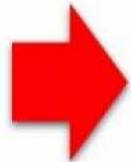
Clinical Picture



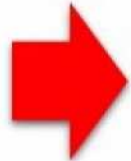
- **Weakness**
- **Jaundice**
- **Hypogonadism**
- **Palmar erythema**
- **Spider nevi**
- **Fetor hepaticas**



Liver cell failure

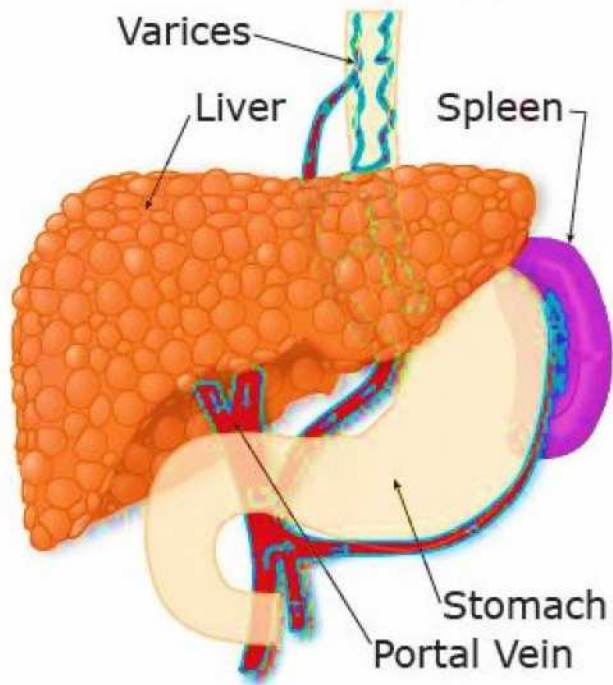


**Portal
hypertension**

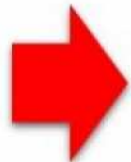


Hepatoma

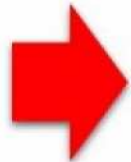
Clinical Picture



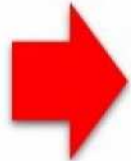
- ① **Esophageal varices leading to Hematemesis**
- ② **Splenomegaly**
- ③ **Ascites**



Liver cell failure

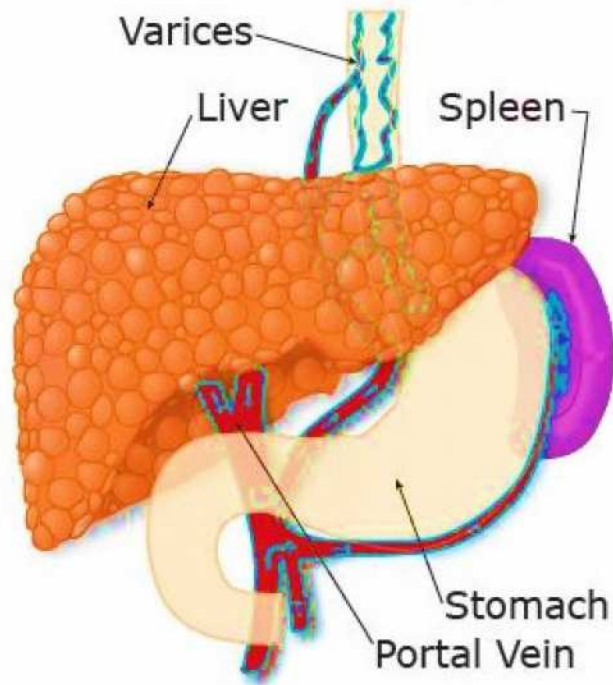


Portal hypertension

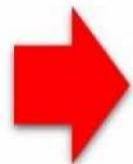


Hepatoma

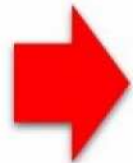
Clinical Picture



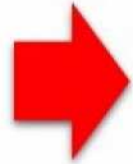
- ① Pain
- ② Sudden deterioration of liver functions
- ③ Rapid expansion of a *cirrhotic* nodule



Liver cell failure



Portal hypertension



Hepatoma

Special Investigations

Abdominal Ultrasonography

Endoscopy

To detect esophageal
varices

LFTs

Elevated bilirubin-
Increased PT- elevated
liver enzymes

Detecting the cause

Liver biopsy

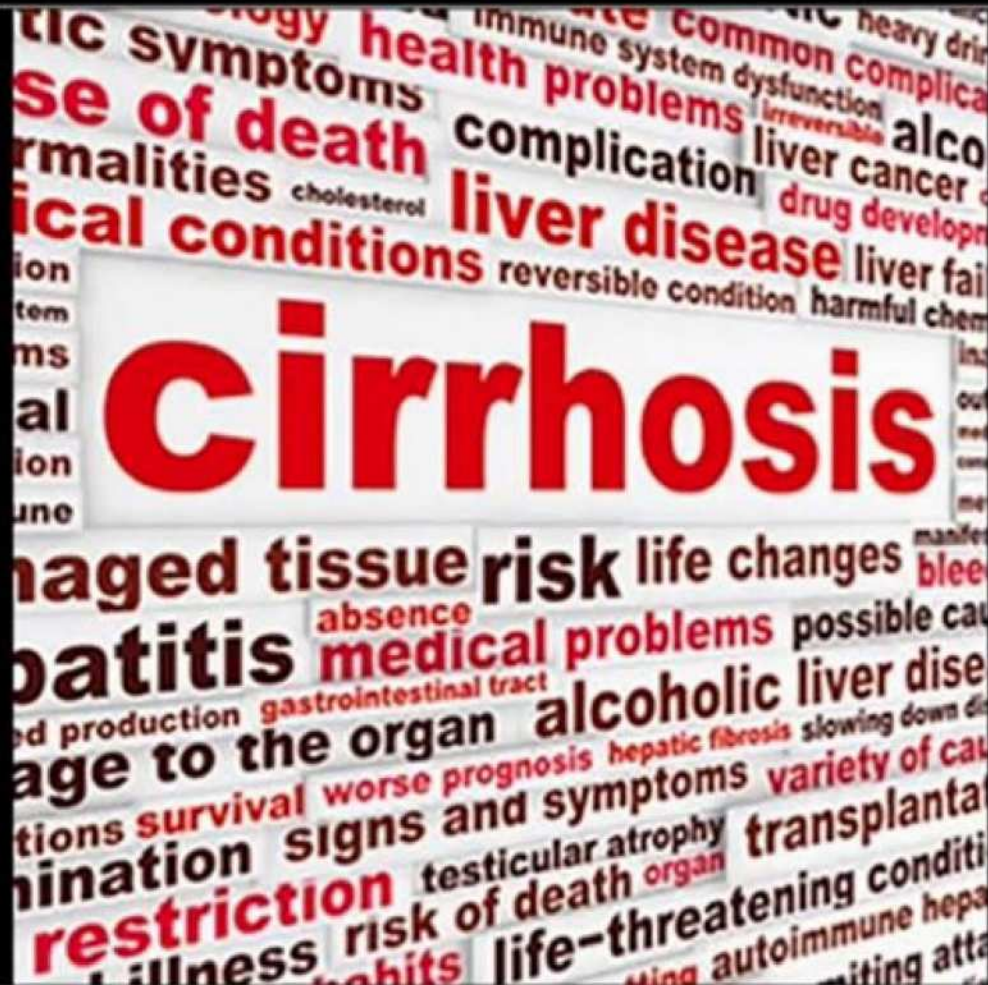
Serology for HBV, HCV
Others,.....



Treatment

- ① **STOP** Alcohol
- ② **Avoid Aspirin** as it aggravates bleeding tendency
- ③ **Adequate and correct nutrition** with vitamin supplements
- ④ **Decrease Sodium intake** to treat ascites
- ⑤ **No specific treatment** is available

Liver Cirrhosis [2]

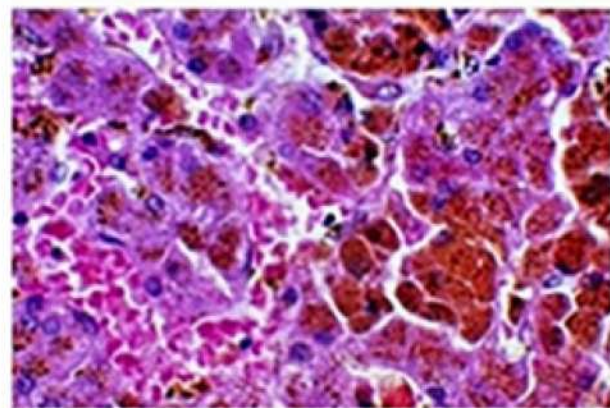


Dr. Tarek Abdelhamid M.D.

Specific causes:

Hemochromatosis

- 1 Excessive **Iron** absorption from the GIT
- 2 Iron deposition in tissues causing:
Skin: Bronzed coloration
Pancreas: DM
Liver: Cirrhosis
Heart: Heart Failure
- 3 Increase plasma iron and serum ferritin
- 4 Elevated hepatic iron content in liver biopsy
- 5 Treatment by Repeated **Venesection** and **Desferoxamine**



Specific causes:

Wilson disease

① Excessive **Copper** deposition in different tissues

Liver: Acute Hepatitis & CAH

Brain: Extrapyrarnidal Manifestations

Eye: KFR

③ **Low** Ceruloplasmin level in blood

④ Elevated hepatic copper content in liver biopsy

⑤ Treatment by **D-Penicillamine**



Specific causes:

Primary Biliary Cirrhosis

1 Autoimmune Disorder

2 Antimitochondrial antibodies

3 Features include:

Hepatomegaly-Fat malabsorption-Pruritus

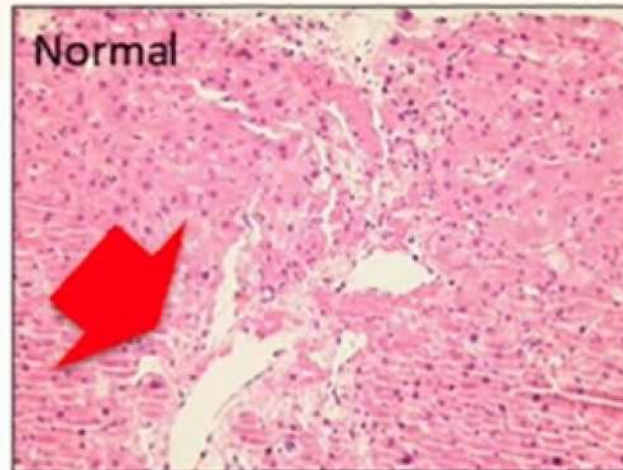
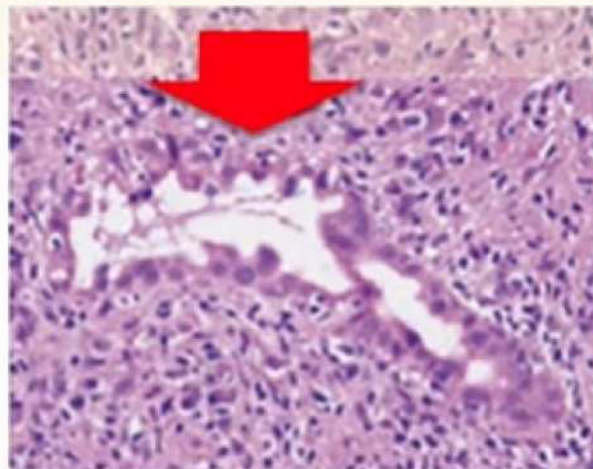
Xanthoma

Painful fingers (Osteomalacia?!)

4 Increased IgM- Cholesterol- SAP

5 Ursodeoxycholic acid- Cholestyramine- Diet

6 Liver transplantation is the only definitive cure

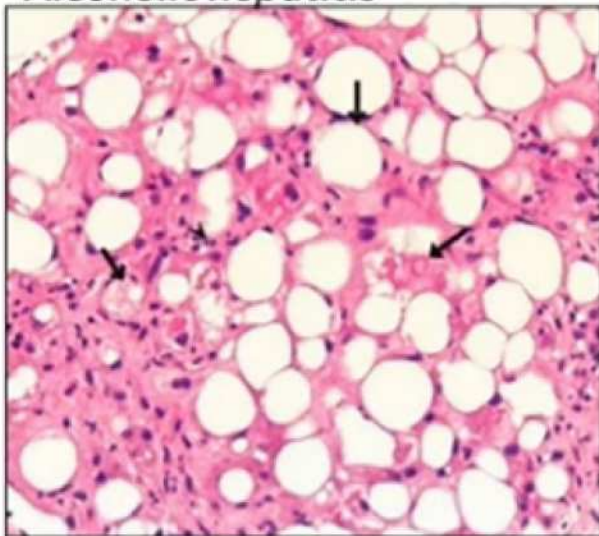


Specific causes:

Alcoholic Cirrhosis

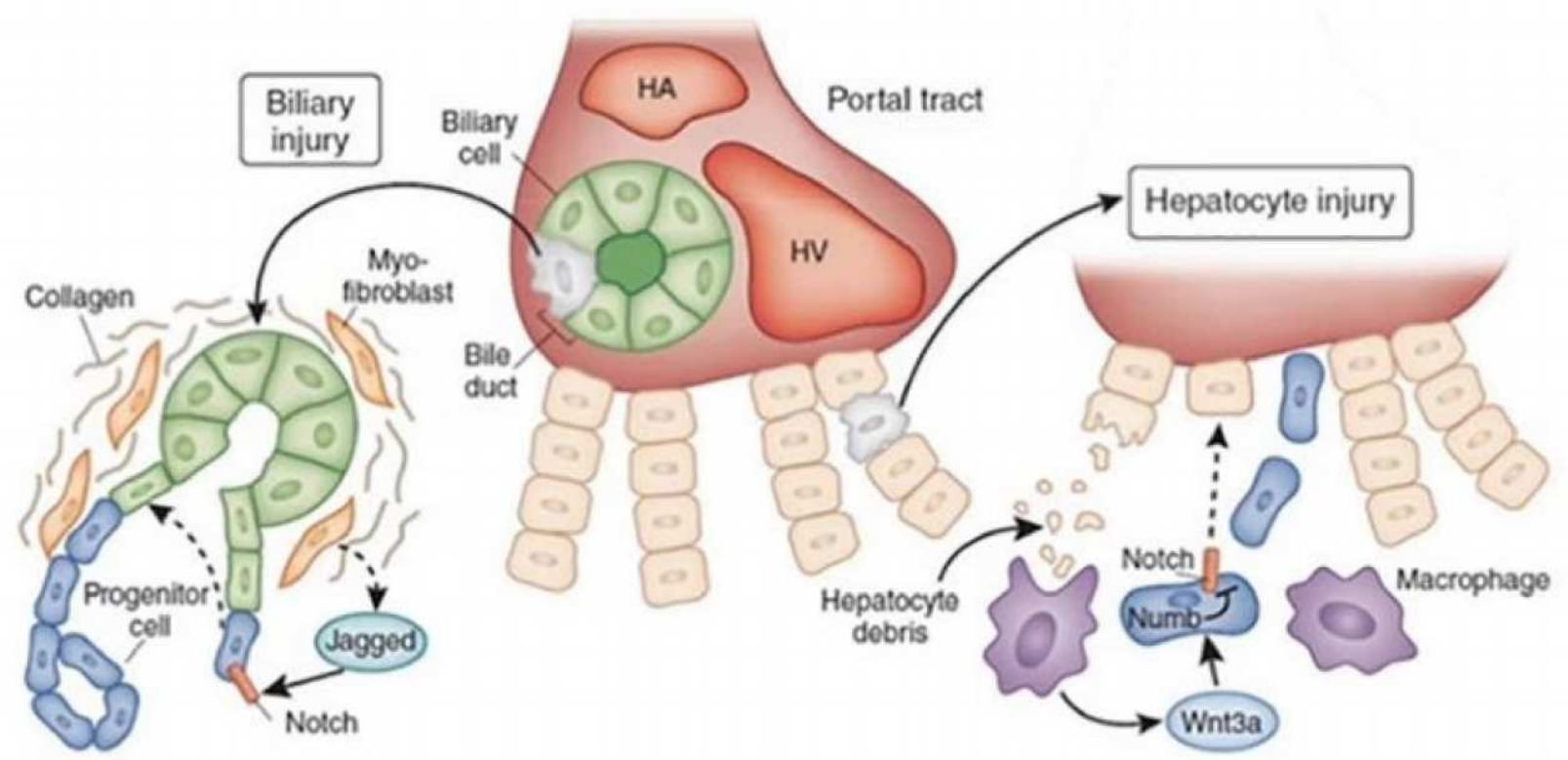
- 1 Excessive Alcohol intake can lead to fatty liver- hepatitis- and Cirrhosis
- 2 **AST > ALT**
- 3 Stopping alcohol is the most important step in the treatment.

Alcoholic hepatitis



AST > ALT

Manifestations of Liver Cell Failure



Mechanism

Metabolic
derangements due
to failure of the
liver to perform its
metabolic functions



Fatigue

Weakness

Mechanism

Inability to
metabolize
bilirubin



Jaundice

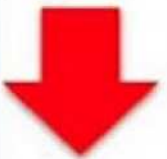


Mechanism

Failure to produce
albumin



Generalized
edema and
ascites



Albumin

Mechanism

Inability to
metabolize
Estrogen



 **Estrogen**



Feminization

In males:

Low frontal hair
Gynecomastia
Impotence
Feminine hair
distribution

Mechanism

Inability to
metabolize
vasodilator
compounds



**Palmar
Erythema**



**Spider
nevi**

Palmar erythema



Spider nevi



Mechanism

Failure to store folic acid: Megaloblastic anemia

Esophageal varices: IDA

Hypersplenism:
Normocytic
Normochromic
anemia

Anemia

Mechanism

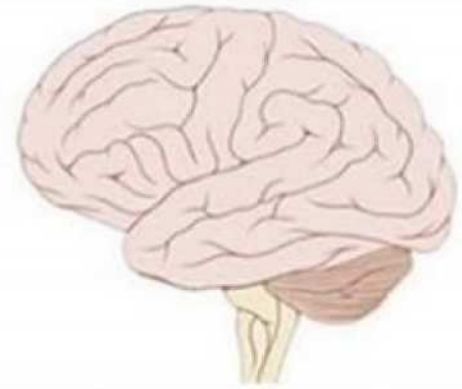
Failure to
metabolize
ammonia



Ammonia Level



**Hepatic
Encephalopathy**



Mechanism

Portal Hypertension

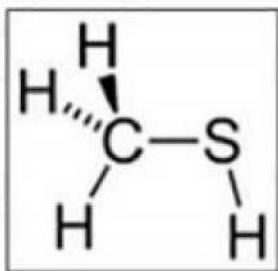


Shunting of blood
from portal to
systemic circulation



Mercaptans content of
amino acid

Methionine (**contains
Sulphur**)



Fetor Hepaticus

Mechanism

Excessive necrosis
of liver cells and
failure to
metabolize
endogenous
pyrogens



Low grade
Fever



Clinical Picture

Encephalopathy

Low frontal hair

Jaundice

Low grade fever

Fetor Hepaticus

Bleeding



Pallor

Gynecomastia

Spider nevi

Ascites *plus*
Generalized
Oedema

IDDM PLUS

Peptic ulceration
(PH)

Palmar
erythema

Hypogonadism

White nails
(low albumin)

Weakness
Muscle wasting
Fatigue

Palmar erythema



White nails



**Med
Learn**

Gastroenterology

You Tube

Acute Hepatic Coma

Causes



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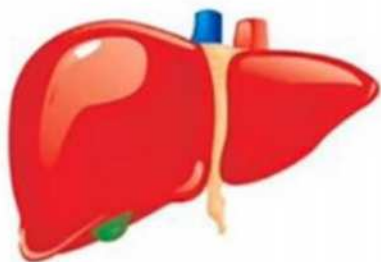
Causes

① Increase
in Ammonia
production

② Hypokalemia

③ Acute
Viral
Hepatitis

⑦ Alcohol
toxicity



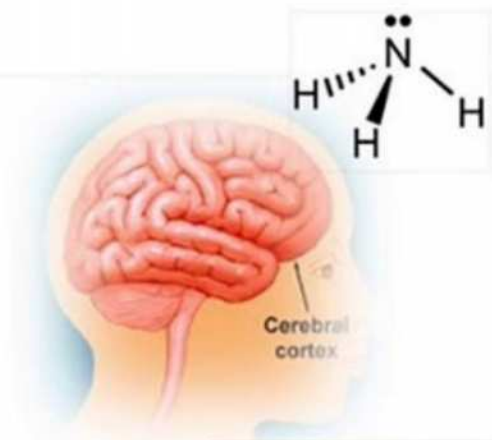
④ Acetaminophen
toxicity

⑥ CNS
depressants
Drugs

⑤ Diminished
cerebral
functions

Increase in Ammonia production

High protein intake
GIT Bleeding
Constipation
Infection
Increased catabolism
e.g. Surgery



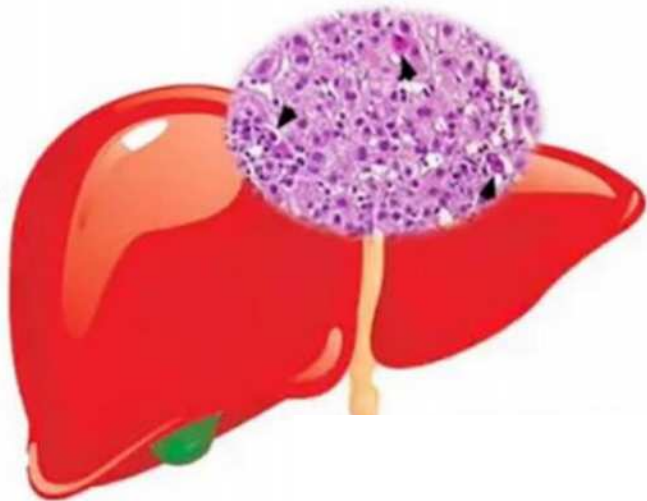
Hypokalemia

Diuretics
Vomiting
Diarrhea
Alkalosis



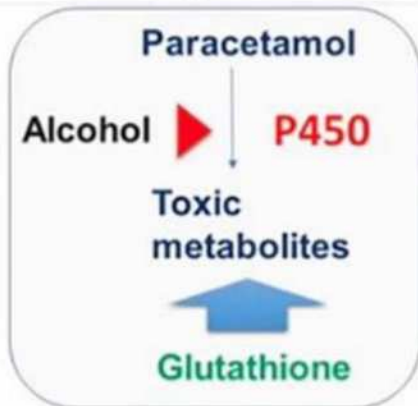
Acute Viral Hepatitis

Acute
hepatic
injury



Acetaminophen toxicity

- ① Paracetamol changes into toxic metabolites by P450
- ② Glutathione binds to these metabolites to detoxify the liver
- ③ This leads to consumption of glutathione which leads to liver toxicity
- ④ Alcohol induces P450 and thus may aggravate the toxicity problem
- ⑤ ttt is by early use of N-Acetyl Cysteine to replenish hepatic glutathione

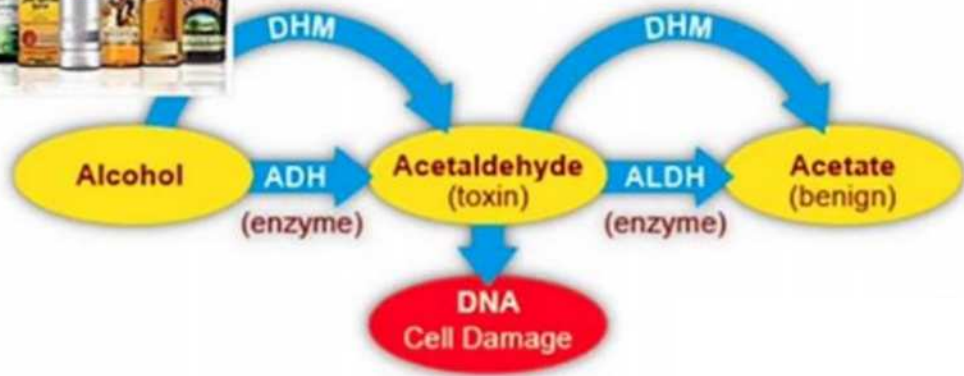


Diminished Cerebral Functions

Hypovolemia



Alcohol toxicity



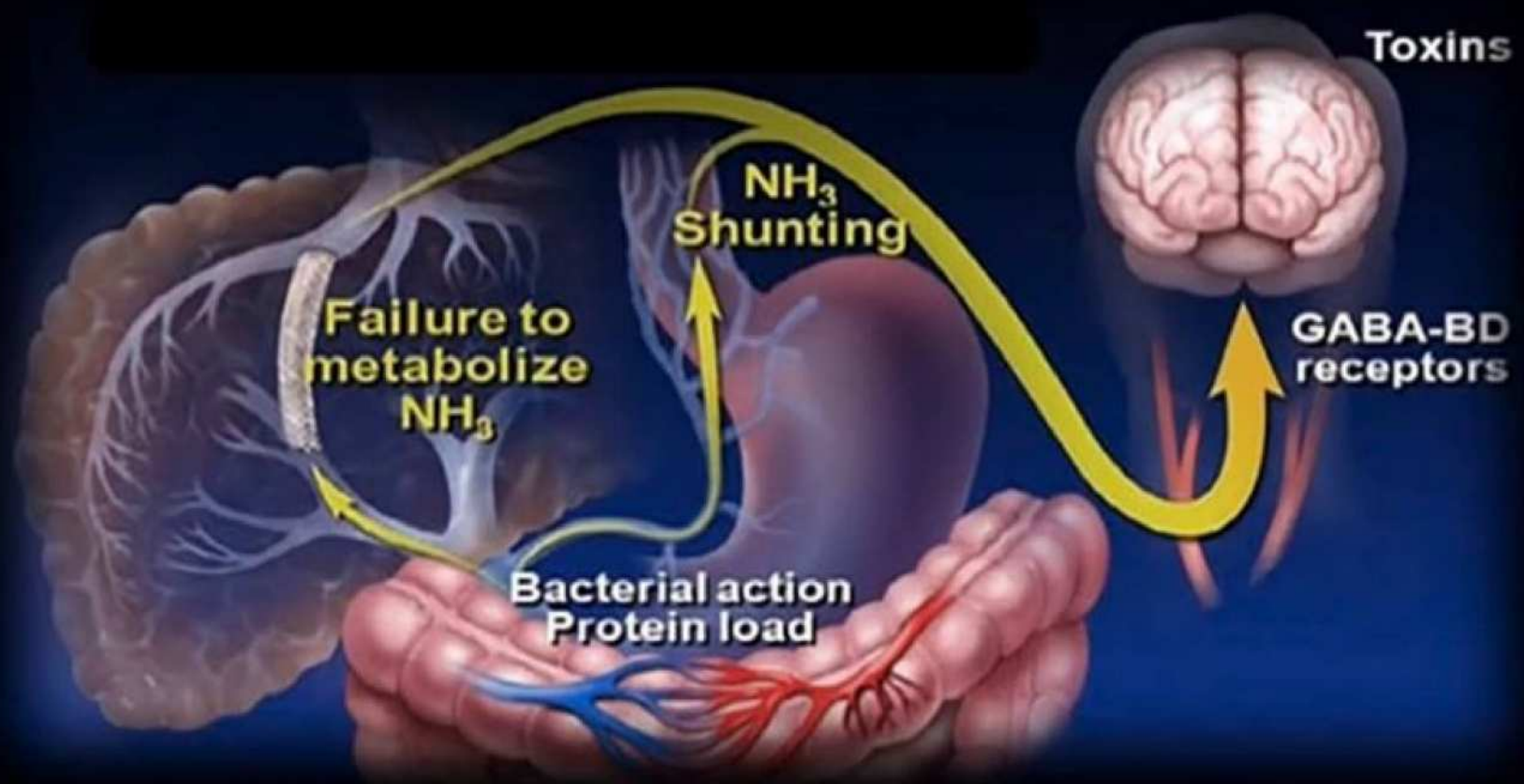
CNS depressants

Narcotics
Sedatives
Tranquilizers



Increased sensitivity of
BNZ Receptors in
patients with LCF makes
them very sensitive to
BNZ.

Hepatic Encephalopathy



Mechanism

Accumulation of
ammonia



**Direct
activation of
GABA
Receptors in
the brain**

Mechanism

Accumulation of
endogenous BNZ



**Brain
Inhibitory
Action**

Mechanism

Accumulation of
endogenous BNZ



**Brain
Inhibitory
Action**

Mechanism

Altered serum
levels of Aromatic
and branched chain
fatty acids



**Production of
false
neurotransmitter
[Octopamine]**

Mechanism

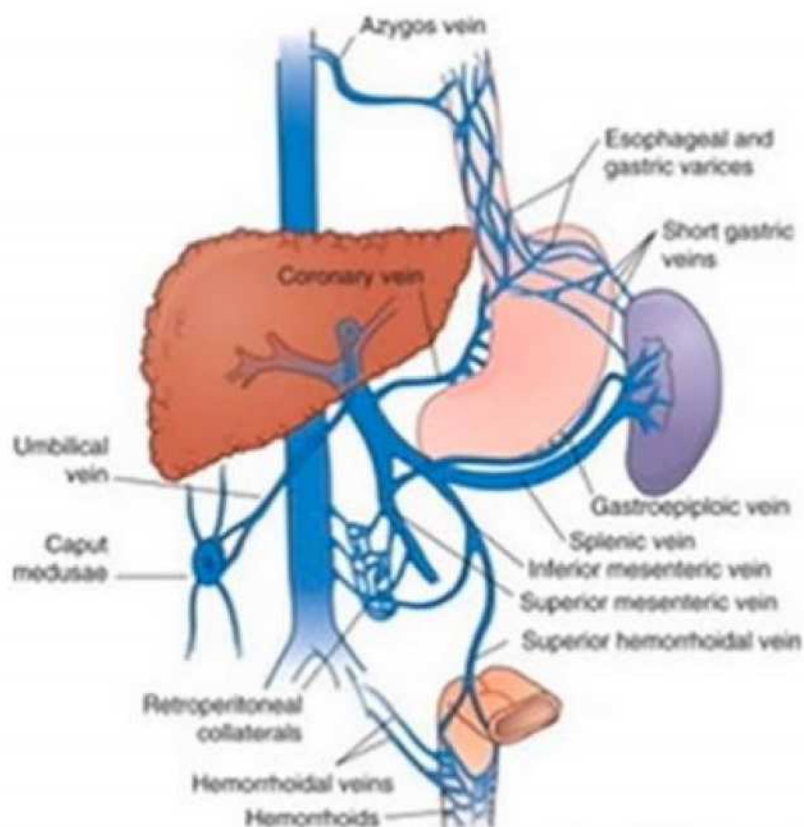
Shunting of compounds such as Mercaptans and short chain fatty acids and Phenols



Reaches systemic circulation (via opened portal collaterals)



Affects brain Functions



Mechanism

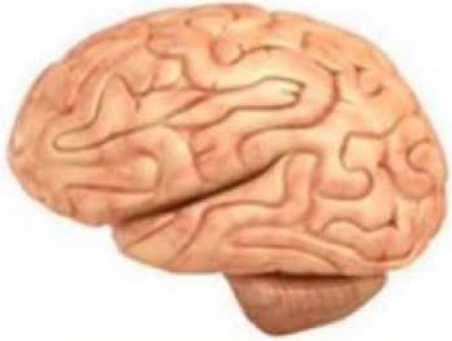
Deposition of
Manganese (**Mn**) in
basal ganglia



**Extrapyramidal
Manifestations**



Clinical picture



Euphoria
Depression
Confusion
Slurred speech



Disturbed
Sleep Rhythm



Asterixis

Investigations

Non specific, however, the presence of **high Ammonia level** in the proper clinical context is highly suggestive of the diagnosis.



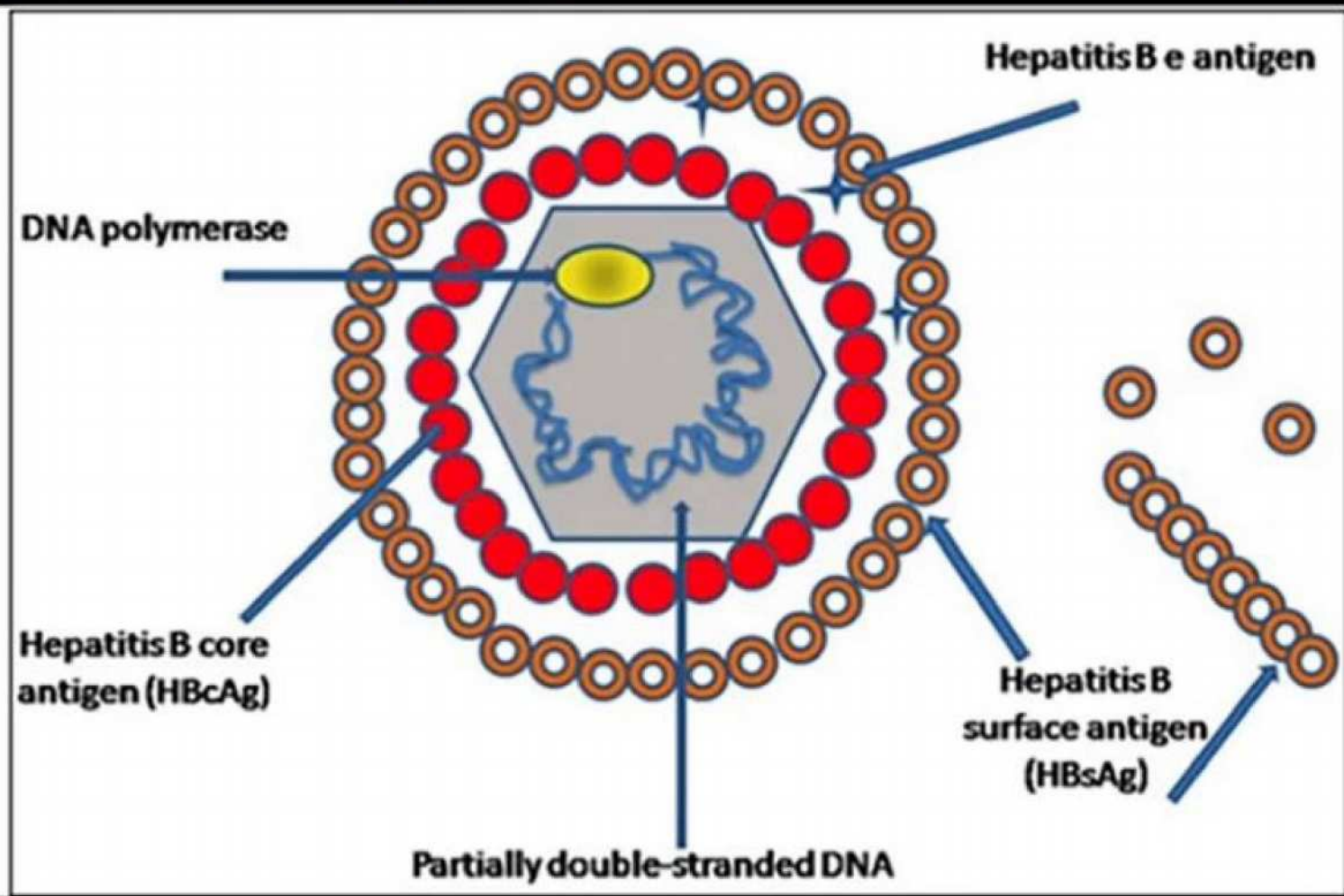
Treatment

- 1 Remove any ppt factor
- 2 Decrease ammonia level 
- 3 Zinc supplements (?!)
- 4 Avoid Opioids -Tranquilizers
- and Sedatives
[If agitated give **Oxazepam**]

Decreasing protein
intake
Laxatives
Enema
Neomycin
Lactulose orally

- 5 **Flumazenil**
(a BNZ Receptor antagonist)
may improve cognitive
functions in some cases).

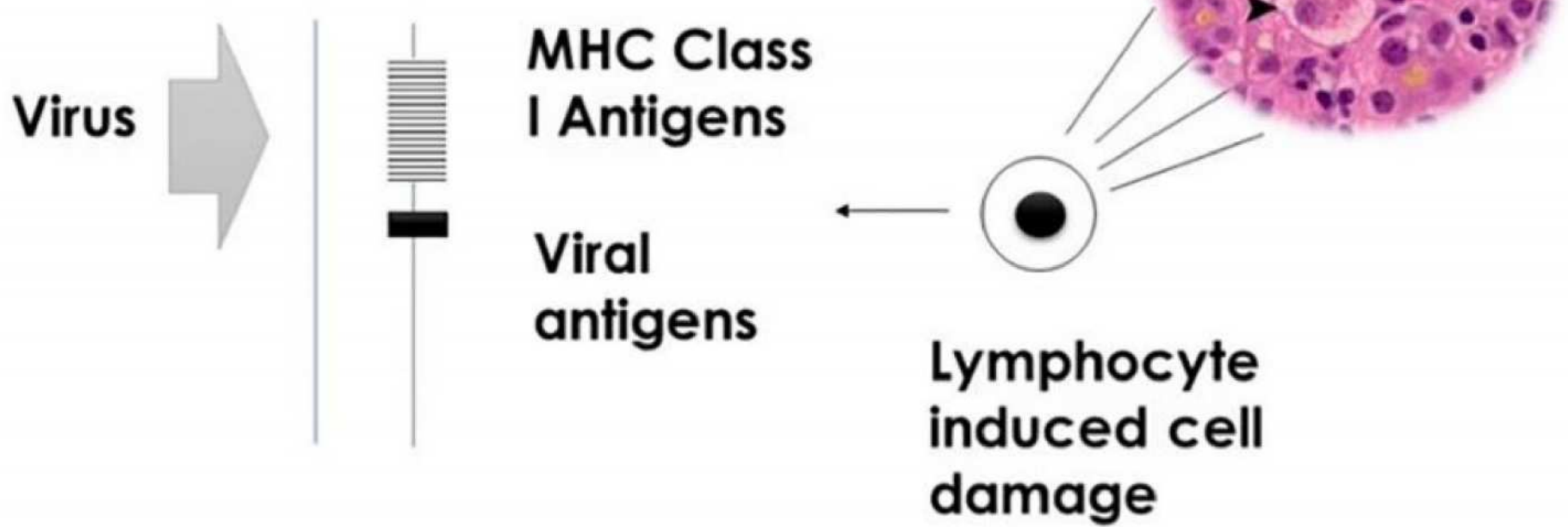
Virus B Hepatitis



Definition

Acute viral infection of the liver cells caused by HBV

Mechanism of injury



Clinical Picture

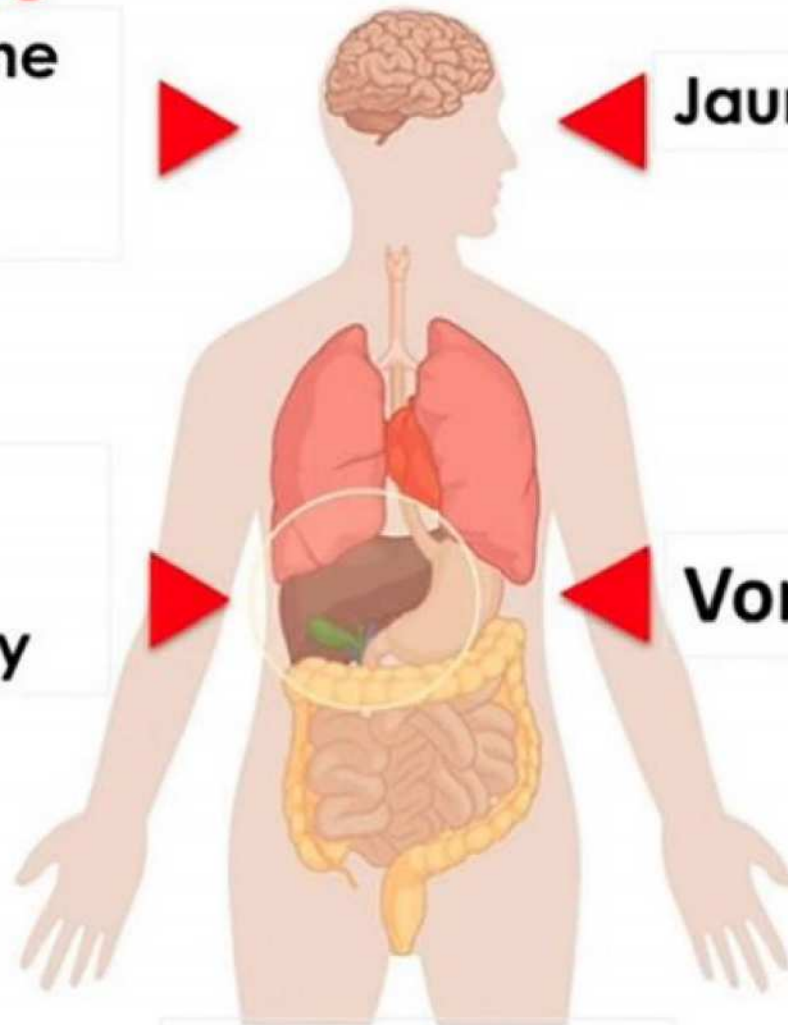
Headache
Fever
malaise

Jaundice

Acute pain
Tender
hepatomegaly

Vomiting

Cure (**Majority**)
Acute liver failure
Chronic hepatitis
Cancer (later on)



Clinical Picture

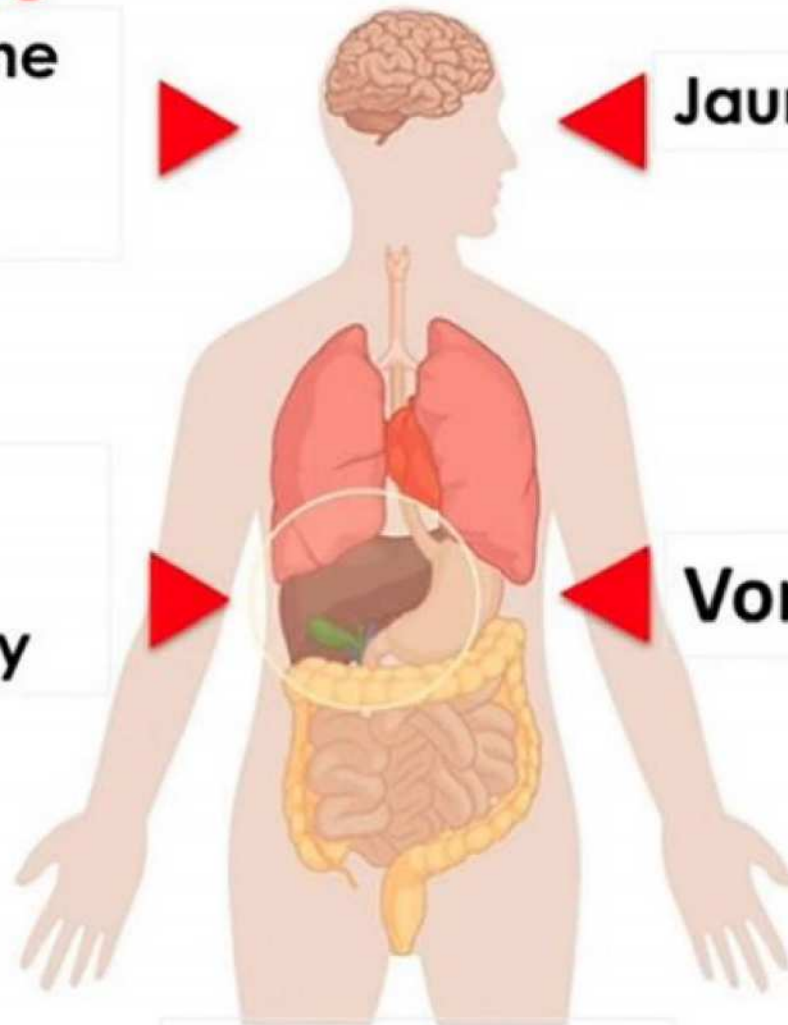
Headache
Fever
malaise

Jaundice

Acute pain
Tender
hepatomegaly

Vomiting

Cure (**Majority**)
Acute liver failure
Chronic hepatitis
Cancer (later on)



Special Investigations

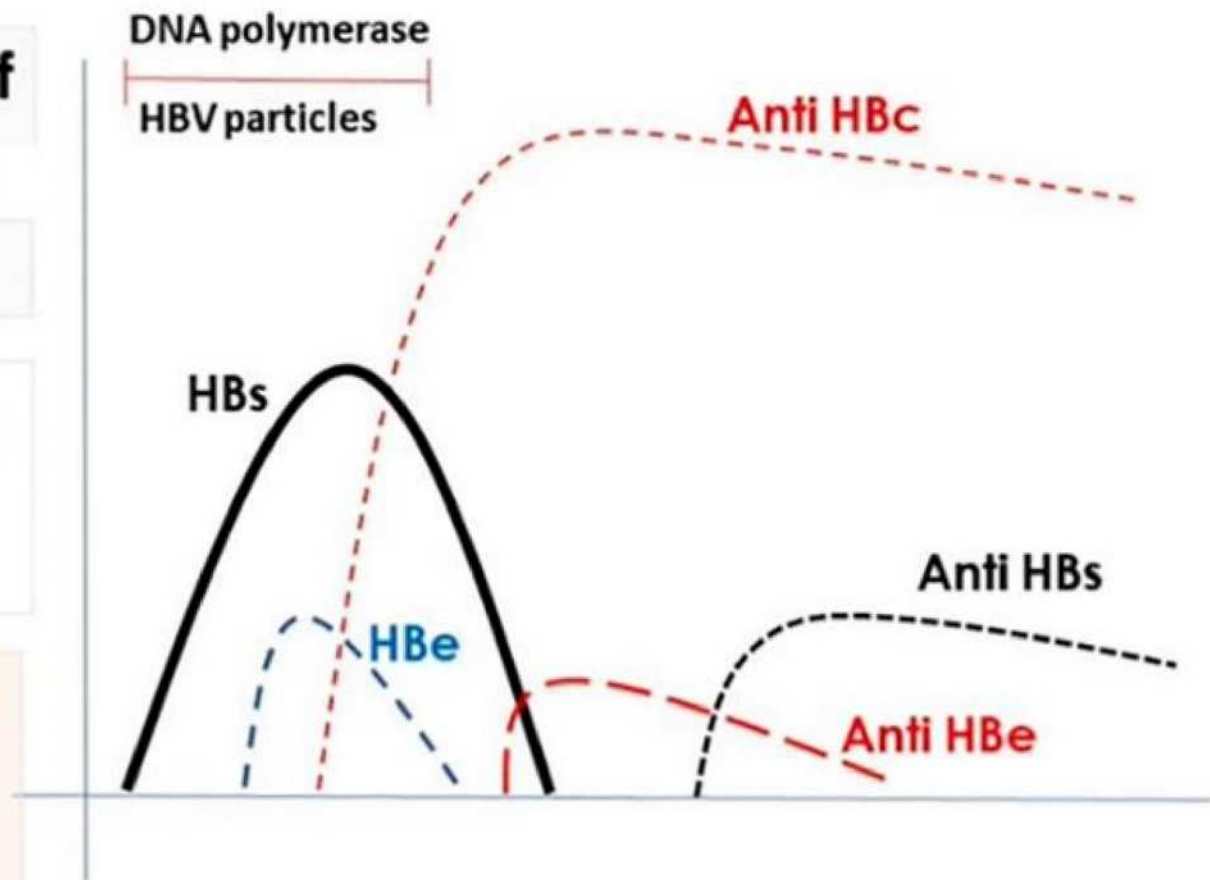
HBs Ag = Presence of the virus (Infection)

Anti HBs = Immunity

Anti HBc = Presence of the virus (Infection)

HBe Ag = active viral replication (high infectivity)

Anti HBe = less infectivity



Treatment

- ① **Bed rest**
- ② **IV glucose 10% if persistent vomiting**
- ③ **Palatable meals as tolerated**
- ④ **Avoid excessive exercise- Alcohol- and Hepatotoxic drugs.**
- ⑤ **HBIG may attenuate severe illness if given within 7 days of exposure**

HBV Vaccination

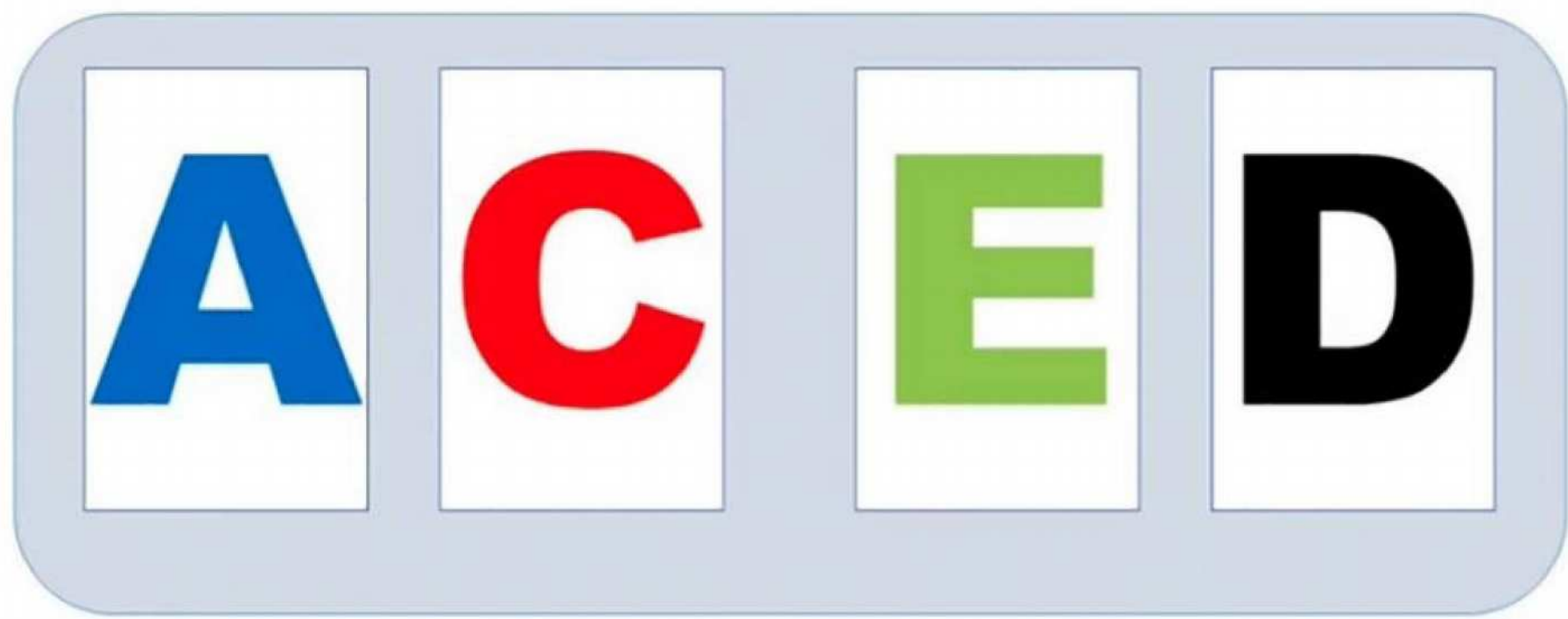
The vaccine is recommended for

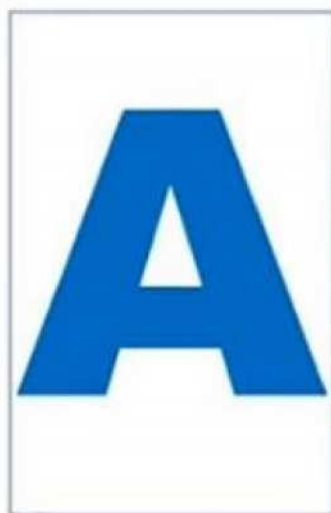
- 1- Multiple anticipated blood transfusions
- 2- Drug users
- 3- Sexual contacts of the patient
- 4- Health care workers

Note: The vaccine is currently recommended for both routine infant & adult vaccination programs

Follow up for the effectiveness of the vaccination is by measuring: **Anti-HBs titers**

Other Types of Viral Hepatitis





- ① Feco oral transmission via food or water
- ② **NO** Chronicity
- ③ **NO** Cancer
- ④ RNA hepatovirus (Picornavirus family)
- ⑤ IgM anti-HAV is an excellent test for Diagnosis
(Note: IgG anti-HAV ONLY indicate Previous Exposure)



- ① High Risk of Chronicity
- ② Detection of anti- HCV antibodies is the best diagnostic test
- ③ (Unlike HBV)- the **Anti HCV is NOT protective**
- ④ Parenteral transmission is the most common form of transmission



E



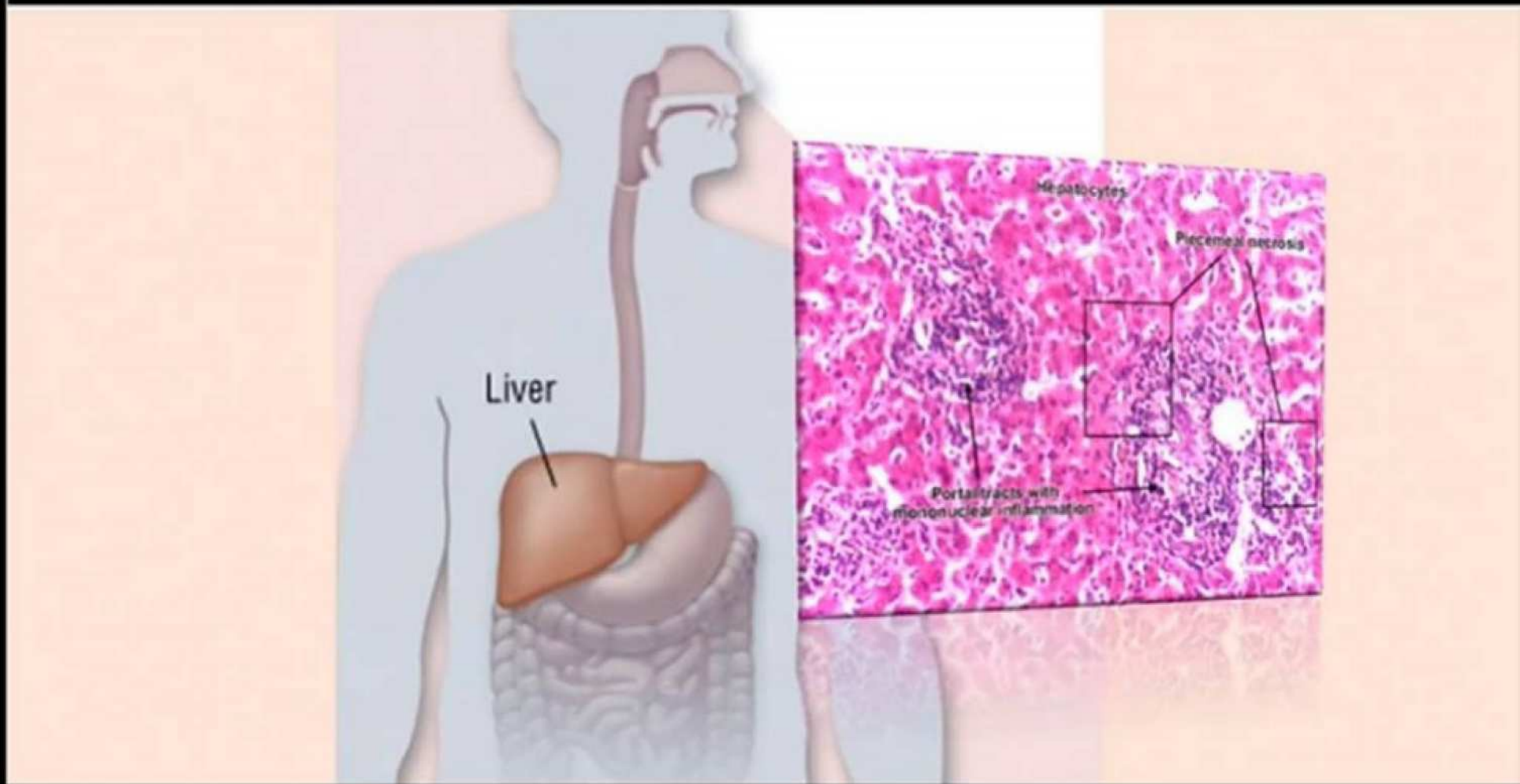
- ① Responsible for water borne viral hepatitis in India, Afghanistan, and Mexico.
- ② Should be considered in any patient who develops acute hepatitis after a trip to an endemic area.
- ③ **NO** Chronicity.
- ④ High Mortality in Pregnant Women
- ⑤ Elevated IgM Anti HEV aids the diagnosis

D



- ① Defective RNA Virus
- ② Needs the existence of Virus B to become pathogenic.
- ③ May aggravate the acute stage of viral hepatitis leading to Acute Fulminant Hepatic Failure.
- ④ May +++ Chronic hepatitis as well.
- ⑤ Anti-HDV antibodies are the Most diagnostic serological test.
- ⑥ Best prevented by preventing VBH via the use of HBV vaccine (Anti-HBs)

Chronic Active Hepatitis



Definition

CAH is a chronic inflammatory reaction of the liver (**More than 6 months**) associated with persistently elevated liver enzymes and documented evidence of inflammatory necrosis on liver biopsy.

Aetiology

- 1- Autoimmune "Lupoid hepatitis"
- 2- Post Viral Hepatitis esp. HBV, HCV, HDV
- 3- Drugs e.g. INH and Methyldopa
- 4- Wilson Disease
- 5- Alpha 1 Antitrypsin Deficiency

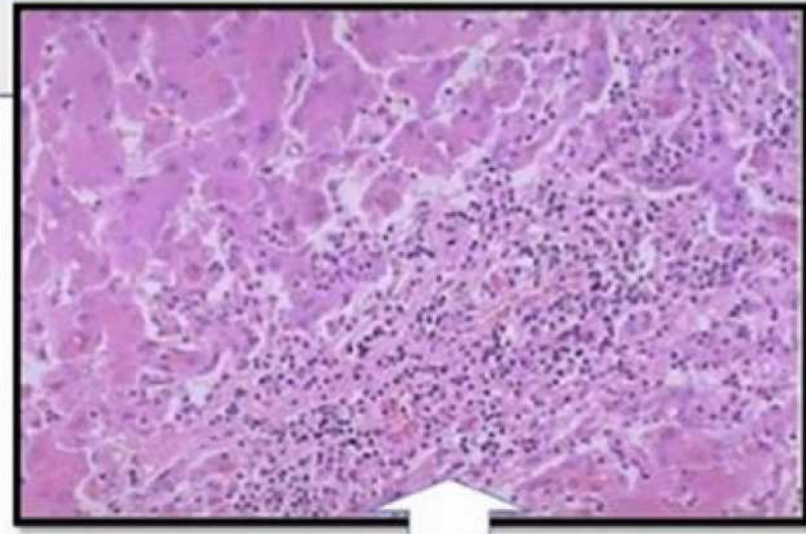
Clinical Picture

Manifestations of the condition may range from asymptomatic patients with persistent elevation of liver enzymes to liver cirrhosis with stigmata of Liver Cell Failure (LCF) such as Weakness- Jaundice- Feminization- bleeding- (See More: **LCF**).

Types

Post Viral

- Males > Females
- Increased incidence of liver malignancy with HBV
- Increased incidence of liver cirrhosis with HCV
- Increased Hbe Antigen and HBV DNA in the early stages of the disease (indicative of active viral replication)



Enlarged portal tract with
mononuclear inflammation
Plus Piecemeal necrosis

Types

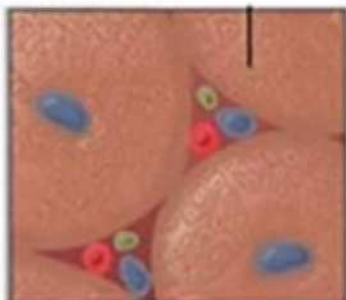
Lupoid Hepatitis

- Typically occurs in young females
- HLA-B8 and DR-3 are often present
- Typical features include Multiple spider nevi, cutaneous striae, acne, hirsutism, and hepatomegaly.
- Increased titer of **Anti-Nuclear Abs** and **Anti smooth muscle Abs**
- May be associated with other autoimmune features e.g. arthritis- nephritis- ulcerative colitis- Thyroiditis



Investigations

Normal Liver



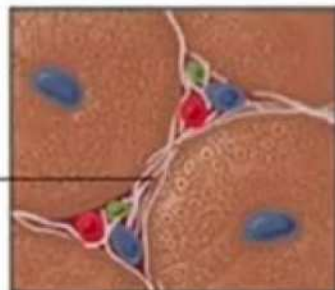
Stage 1 Inflammation, scar tissue begins to form

Scar tissue



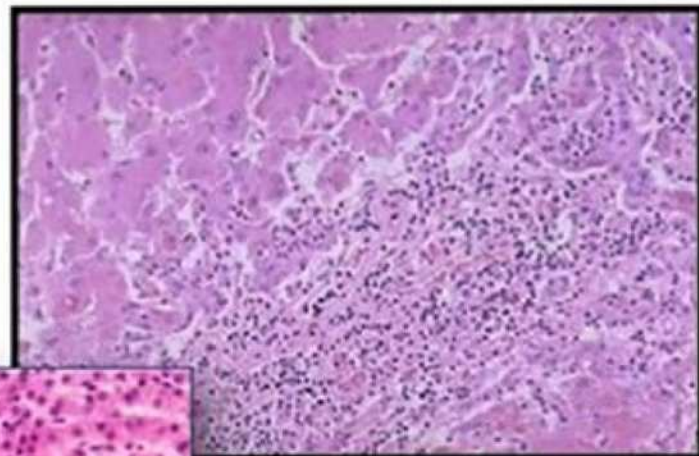
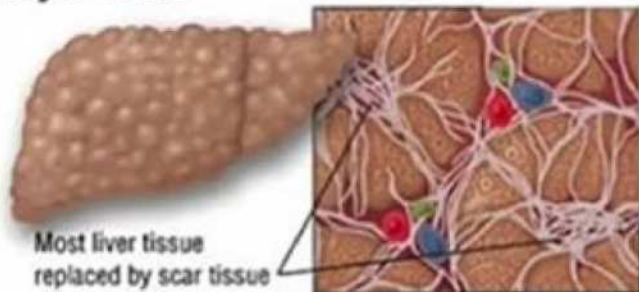
Stage 2

Scar tissue
builds up

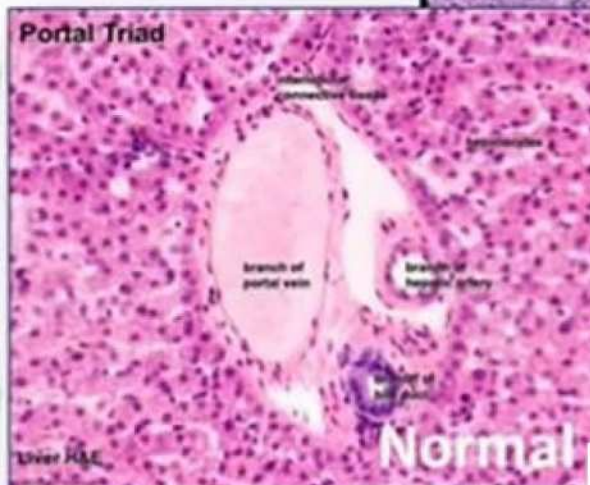


Stage 3 - cirrhosis

Most liver tissue
replaced by scar tissue

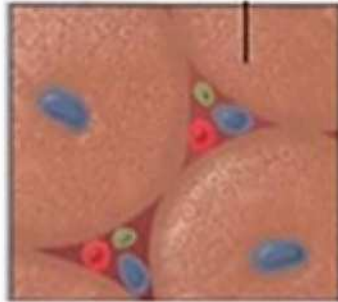


Post Viral
(CAH)



Investigations

Normal Liver



Stage 1

Inflammation, scar tissue begins to form

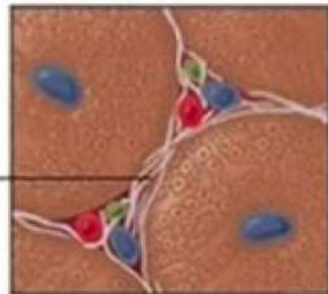


Scar tissue



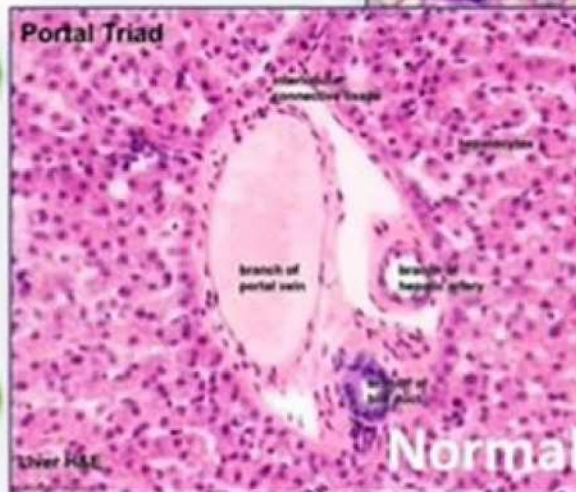
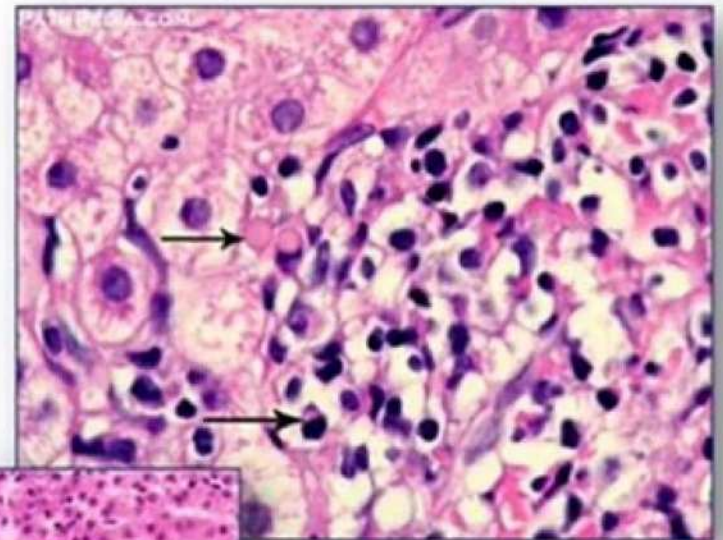
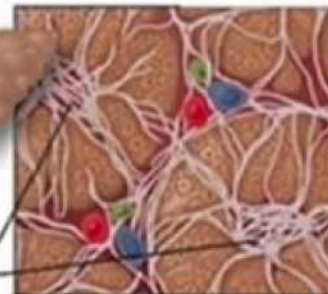
Stage 2

Scar tissue builds up



Stage 3 - cirrhosis

Most liver tissue replaced by scar tissue



Autoimmune hepatitis
(+++ plasma cells)

Treatment

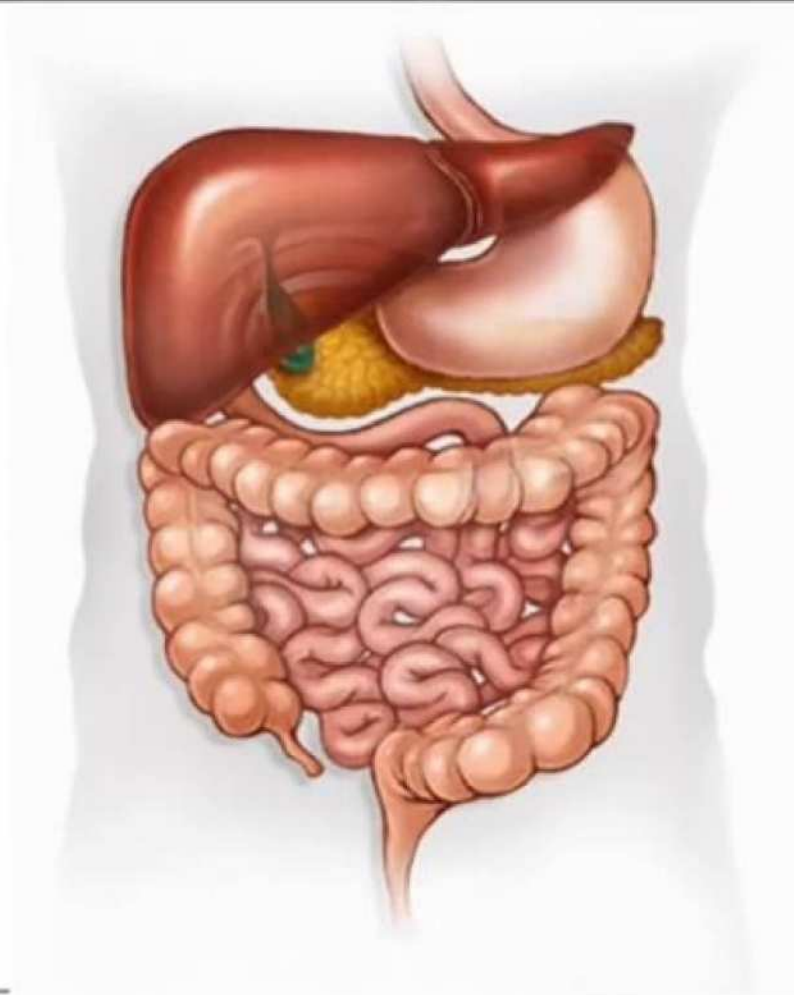
Post Viral:

**Treatment is mainly by
Anti viral therapy.**

Lupoid hepatitis

**Dramatic response to
Corticosteroids (with or
without Azathioprine).**

Malabsorption Syndrome



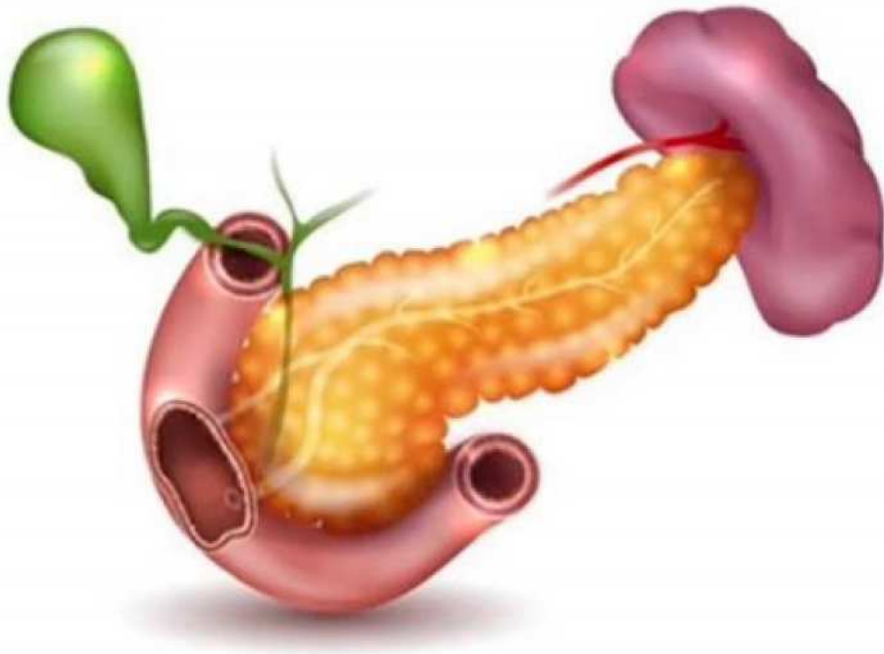
Dr. Tarek Abdelhamid M.D.

Definition

Inability to absorb one ore more nutrients.

Aetiology

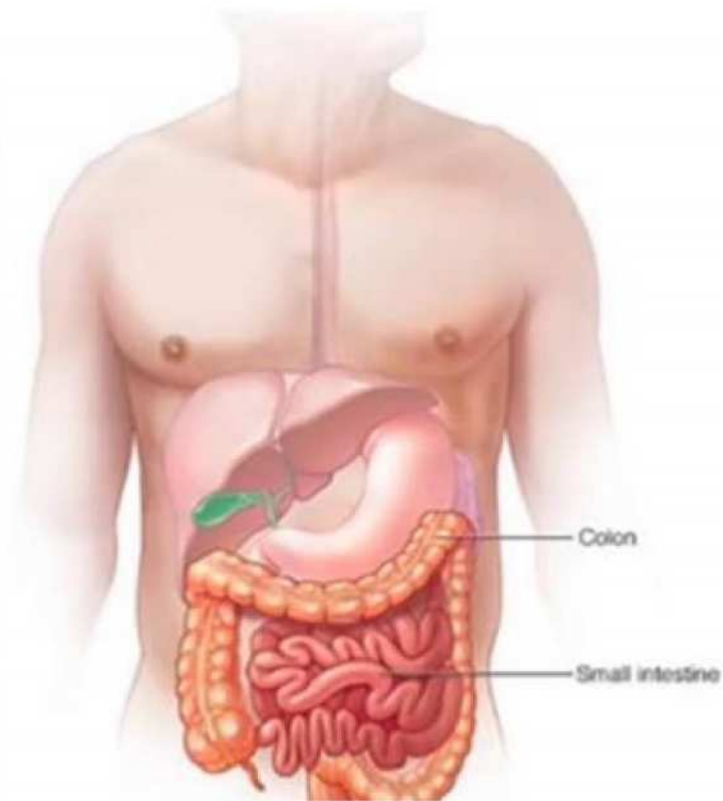
① Deficiency of pancreatic enzymes e.g. Chronic Pancreatitis



Aetiology

② Bile Salts deficiency:

- 1- Obstruction of the Common Bile Duct [CBD]**
- 2- Diseases of the terminal ileum
e.g. T.B.- Crohn's Disease –
Resection**
- 3- Drugs that interfere with EHC of
bile salts e.g. Cholestyramine**
- 4- Increase utilization of bile salts by
bacteria e.g. Blind loop Syndrome
(after surgery- DM- and
Scleroderma)**



Aetiology

③ Intestinal Causes:

1- Resection

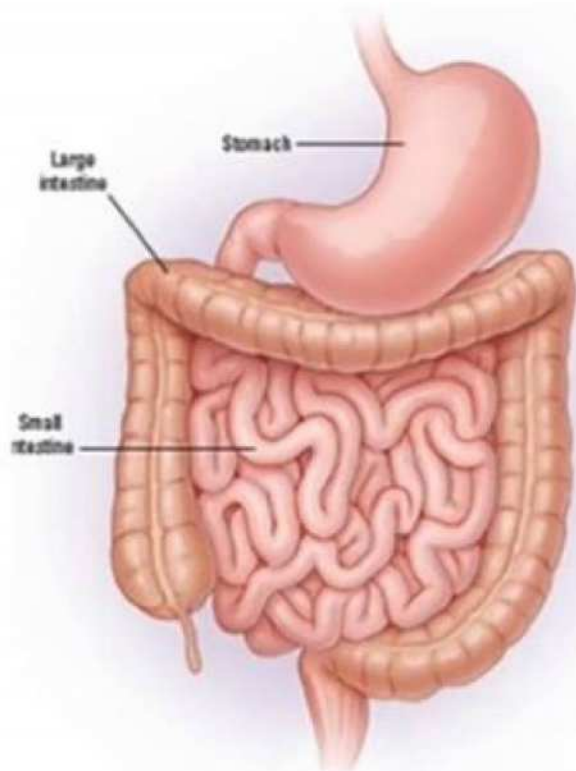
2- Infiltration by lymphoma cells

3- Infection e.g. T.B.

4- Decreased transient time e.g.

Chronic Diarrheal situations:

Infections & Allergic gastroenteritis



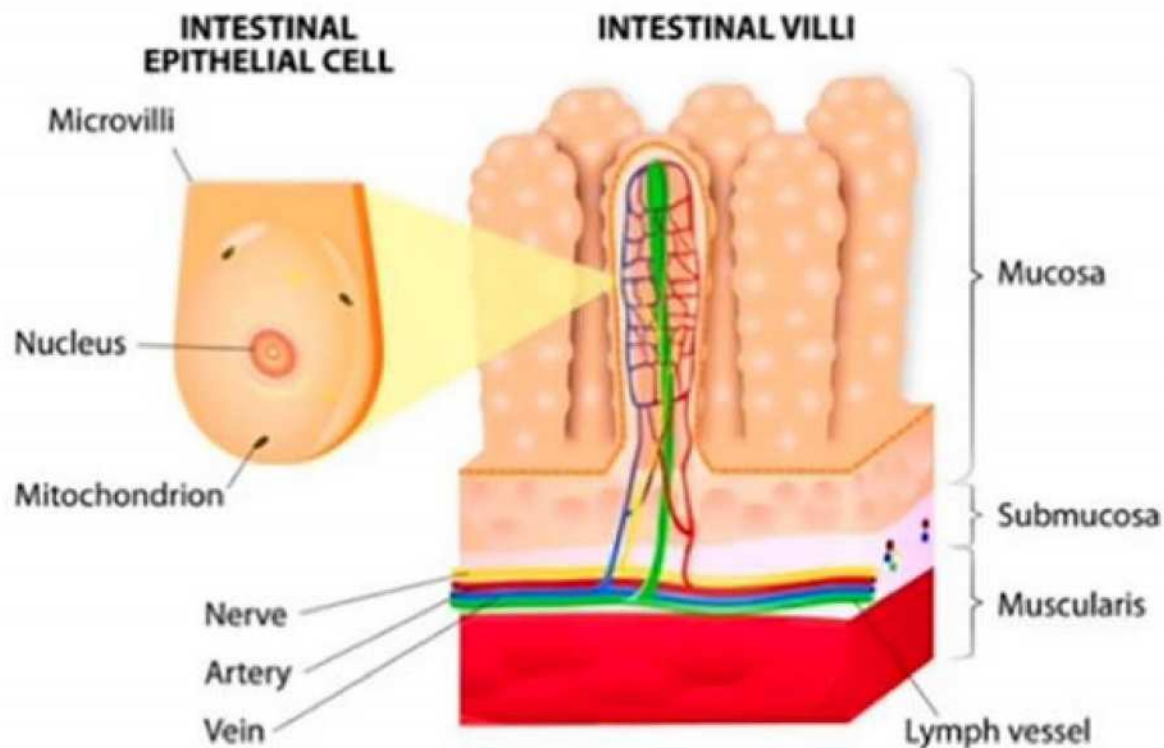
Aetiology

4 Vascular causes:

Arteries: Mesenteric Insufficiency

Veins: or PH

Lymphatics: Lymphangiectasia



Aetiology

5 Other causes for MAS:

Coeliac Disease
(Gluten Enteropathy)

Whipple Disease

Investigations

D-Xylose absorption test

Decrease absorption of D-Xylose and thus decreased its excretion in urine occurs in small intestinal diseases.

Investigations

D-Xylose absorption test

Decrease absorption of D-Xylose and thus decreased its excretion in urine occurs in small intestinal diseases.

Investigations

Small Intestinal Biopsy

To detect the cause of the problem if D- Xylose absorption test is abnormal

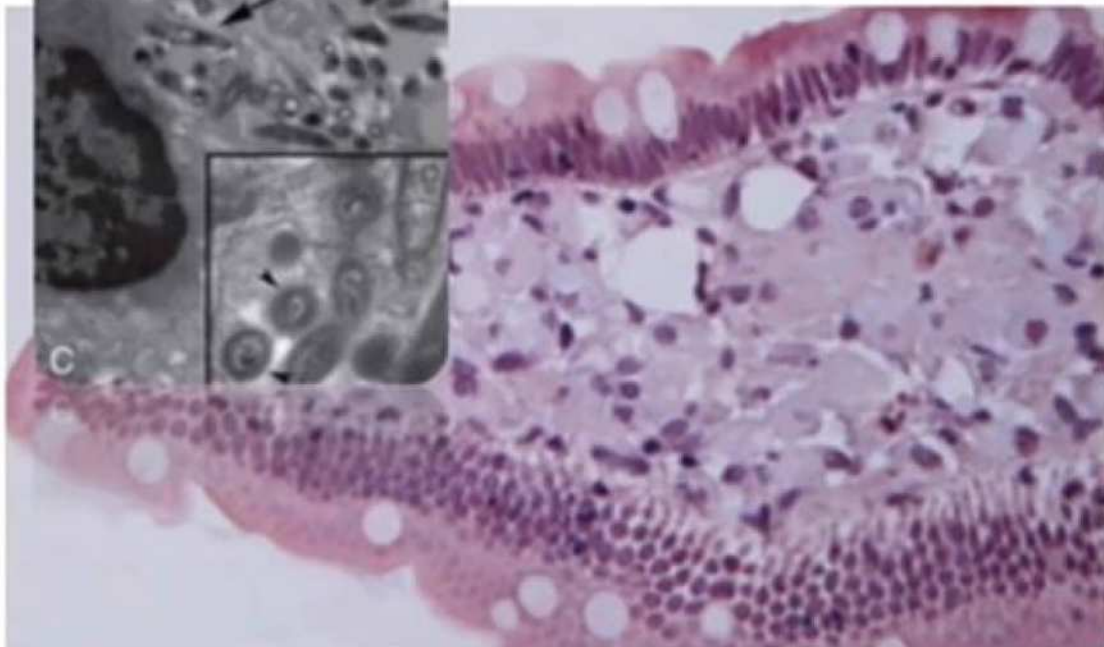
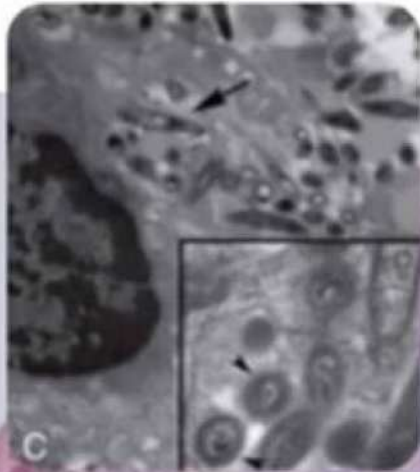


Investigations

Small Intestinal Biopsy

To detect the cause of the problem
if D- Xylose absorption test is
abnormal

PAS +ve Macrophages with bacilli in Whipple Disease

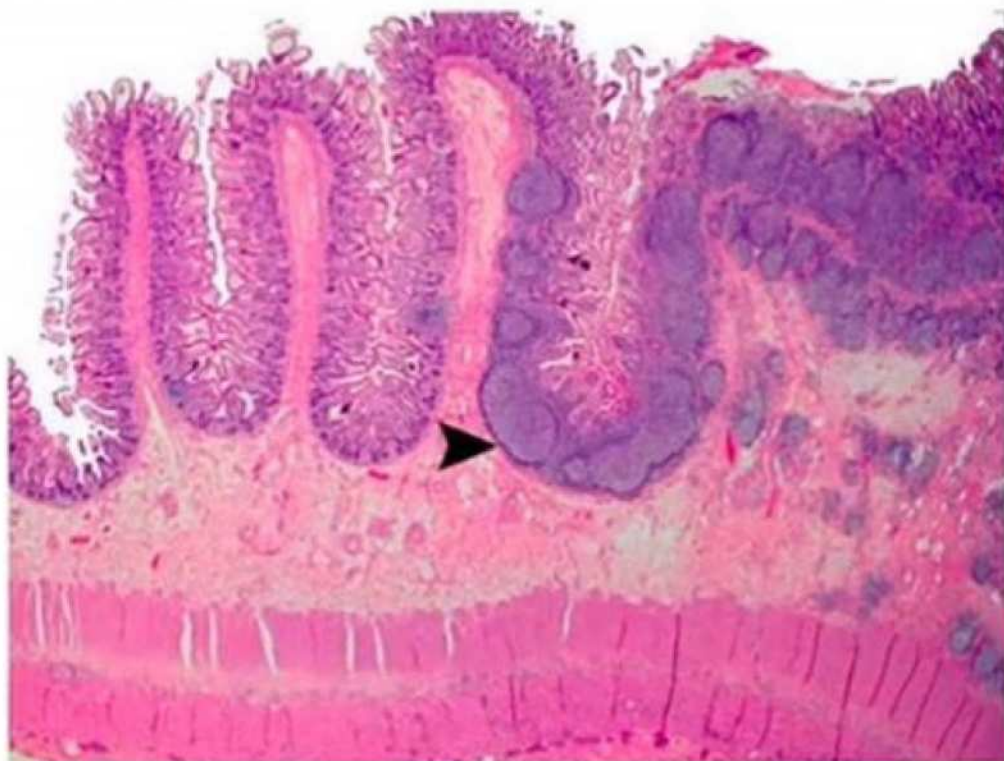


Investigations

Small Intestinal Biopsy

To detect the cause of the problem if D- Xylose absorption test is abnormal

Malignant cells in Intestinal Lymphoma

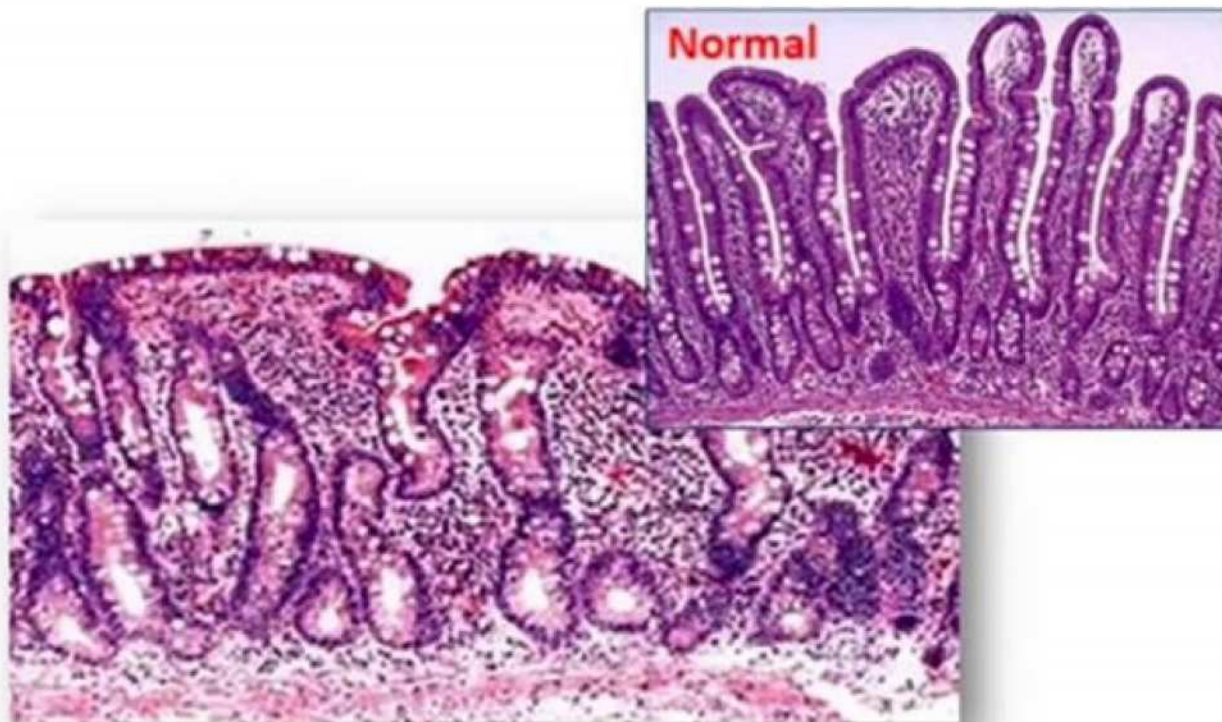


Investigations

Small Intestinal Biopsy

To detect the cause of the problem if D- Xylose absorption test is abnormal

Atrophic villi in Coeliac Disease

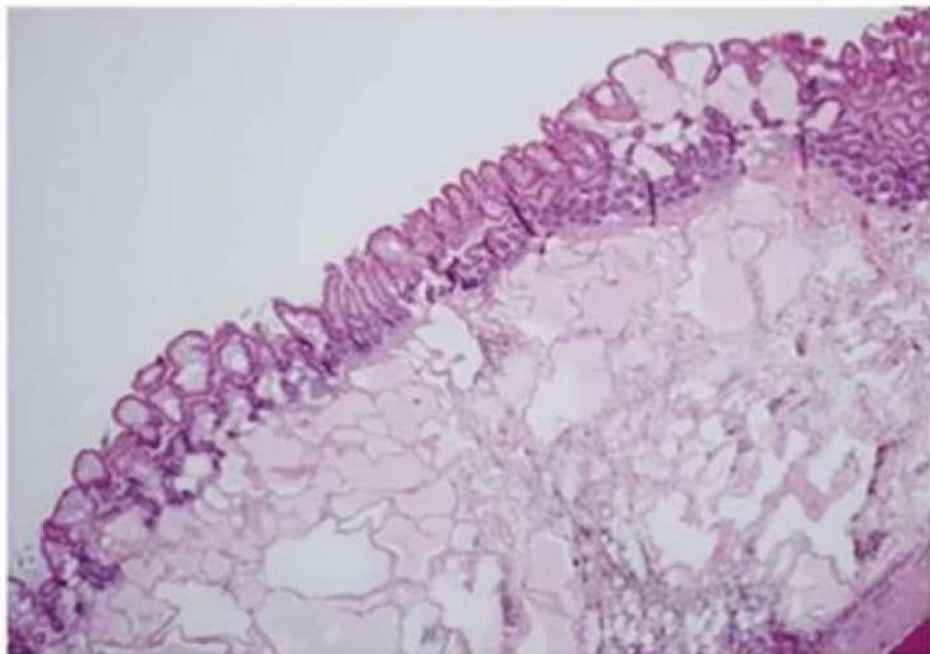


Investigations

Small Intestinal Biopsy

To detect the cause of the problem if D- Xylose absorption test is abnormal

Dilated lymphatics in Intestinal Lymphangiectasia

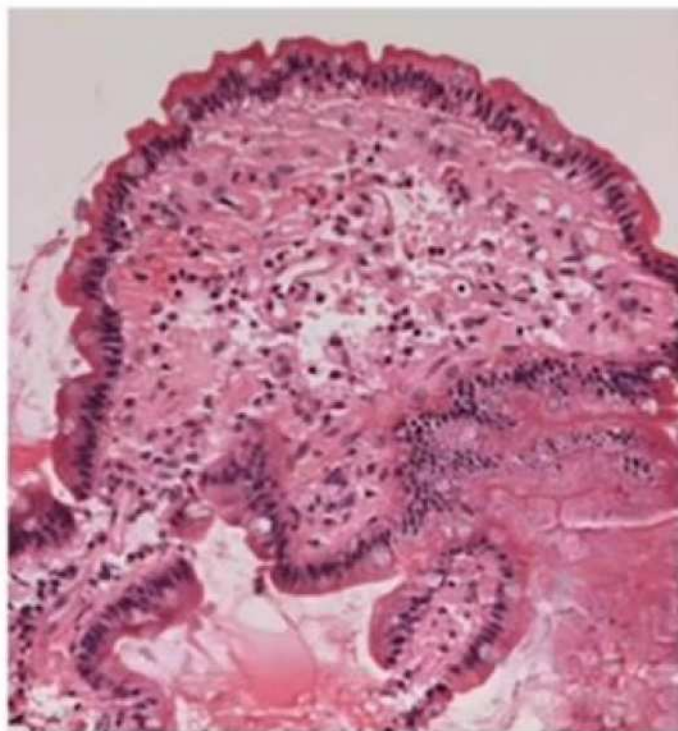


Investigations

Small Intestinal Biopsy

To detect the cause of the problem if D- Xylose absorption test is abnormal

Amyloid material in Amyloidosis

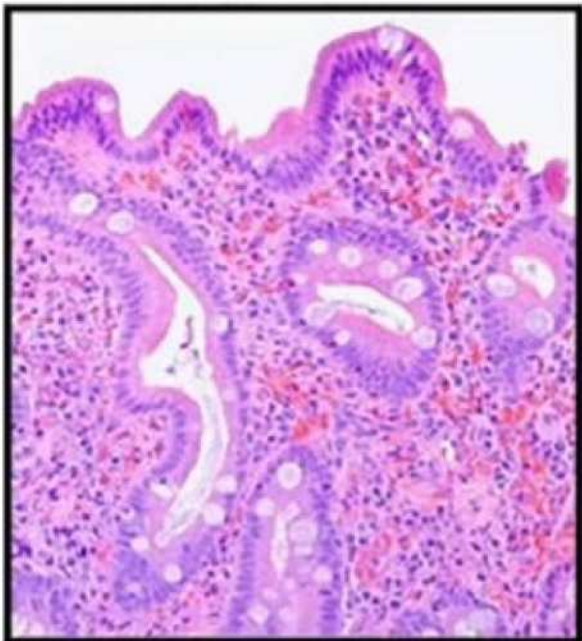


Investigations

Small Intestinal Biopsy

To detect the cause of the problem if D- Xylose absorption test is abnormal

Eosinophilic infiltration in eosinophilic enteritis

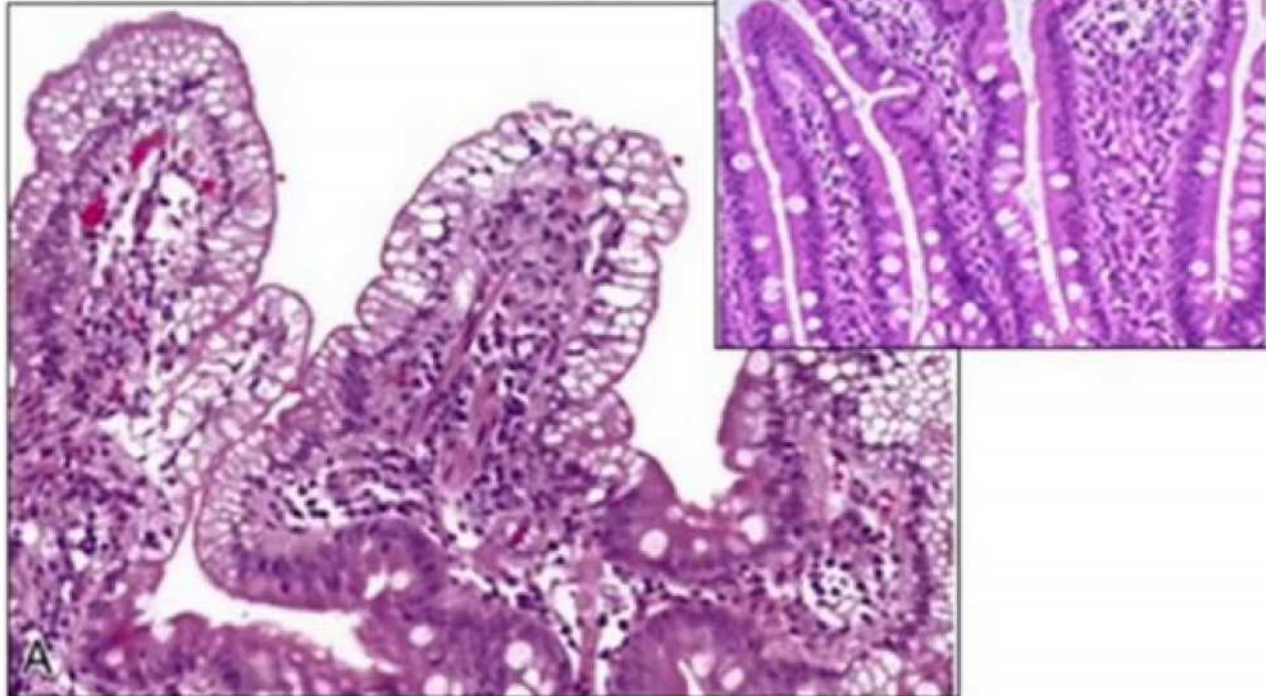


Investigations

Small Intestinal Biopsy

To detect the cause of the problem if D- Xylose absorption test is abnormal

Vacuolated epithelial cell with fat in Abetalipoproteinemia

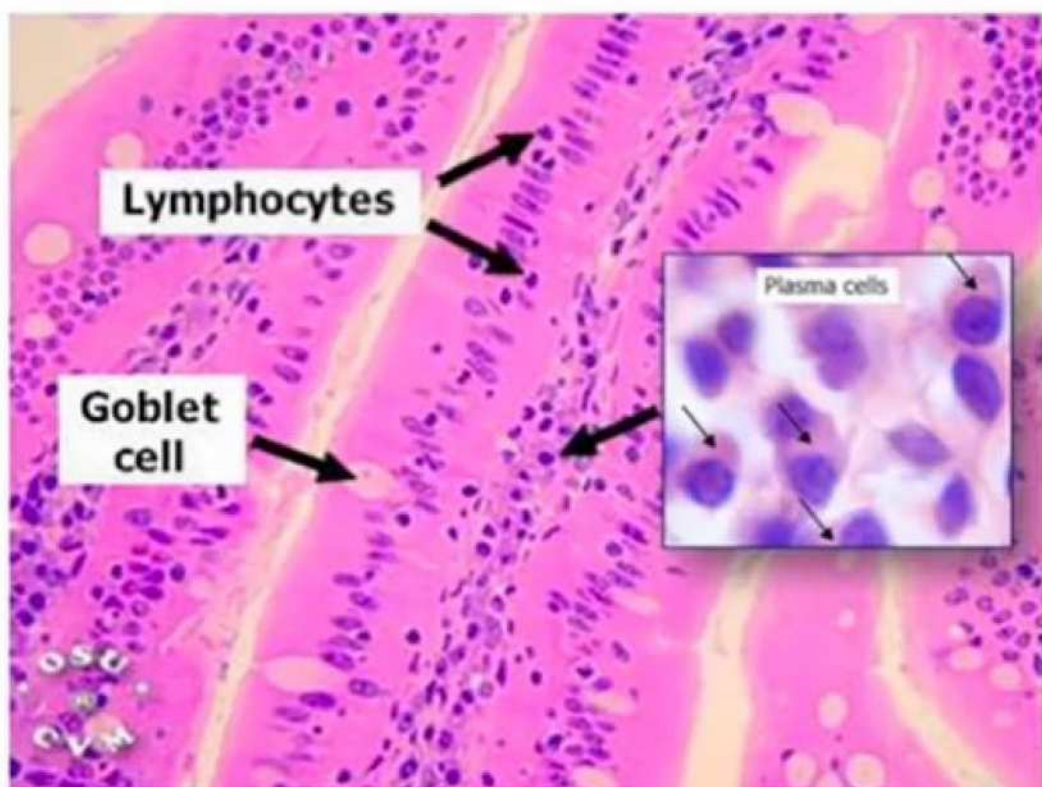


Investigations

Small Intestinal Biopsy

To detect the cause of the problem if D- Xylose absorption test is abnormal

No Plasma cells in Agammaglobulinemia

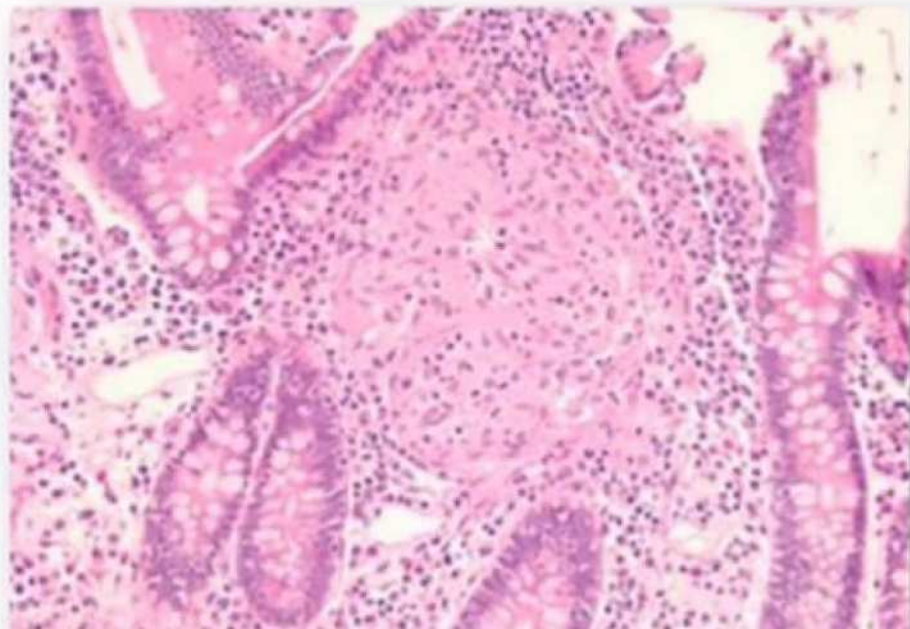


Investigations

Small Intestinal Biopsy

To detect the cause of the problem if D- Xylose absorption test is abnormal

("Non- Caseating") granulomas in Crohn's Disease

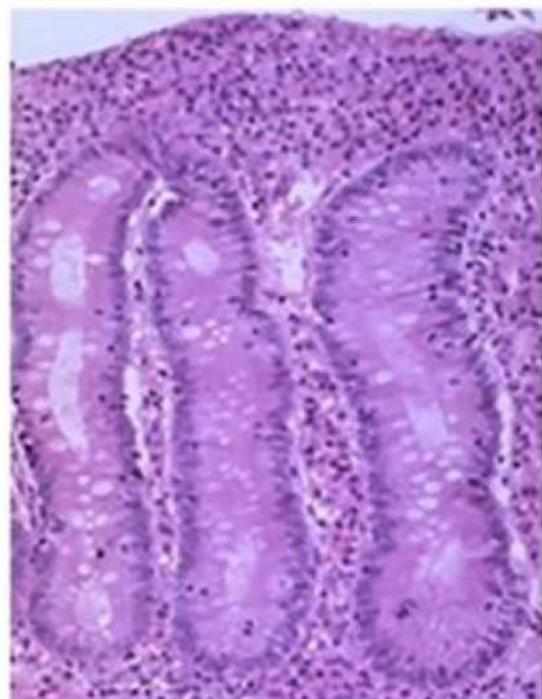


Investigations

Small Intestinal Biopsy

To detect the cause of the problem if D- Xylose absorption test is abnormal e.g.

Patchy damage PLUS Lymphocytic infiltration in Bacterial Overgrowth Syndrome



Investigations

Small Intestinal Biopsy

To detect the cause of the problem if D- Xylose absorption test is abnormal e.g.

Mucosal ulcerations and erosions in ZES

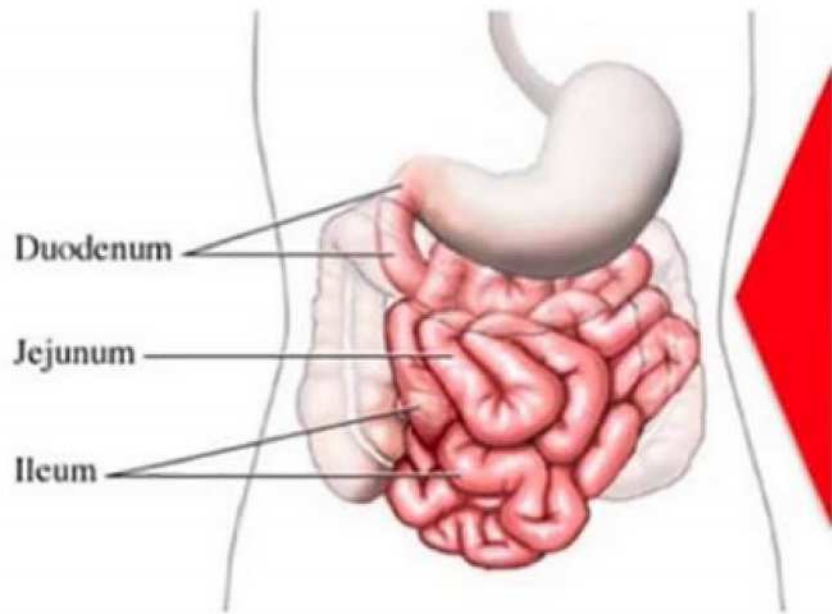
Investigations

Specific investigation

- 1 Calcification at the level of L1 ON X-ray abdomen....Chronic Pancreatitis
- 2 Increased Folate level....Bacterial overgrowth syndrome
- 3 Anti-Saccharomyces Cerevisiae antibodies (ASCA) seem to be associated with Crohn's disease (CD)



Clinical Picture



Chronic diarrhea

Nutritional
deficiency

Flatulence

Loss of weight

Stools float on
toilet water

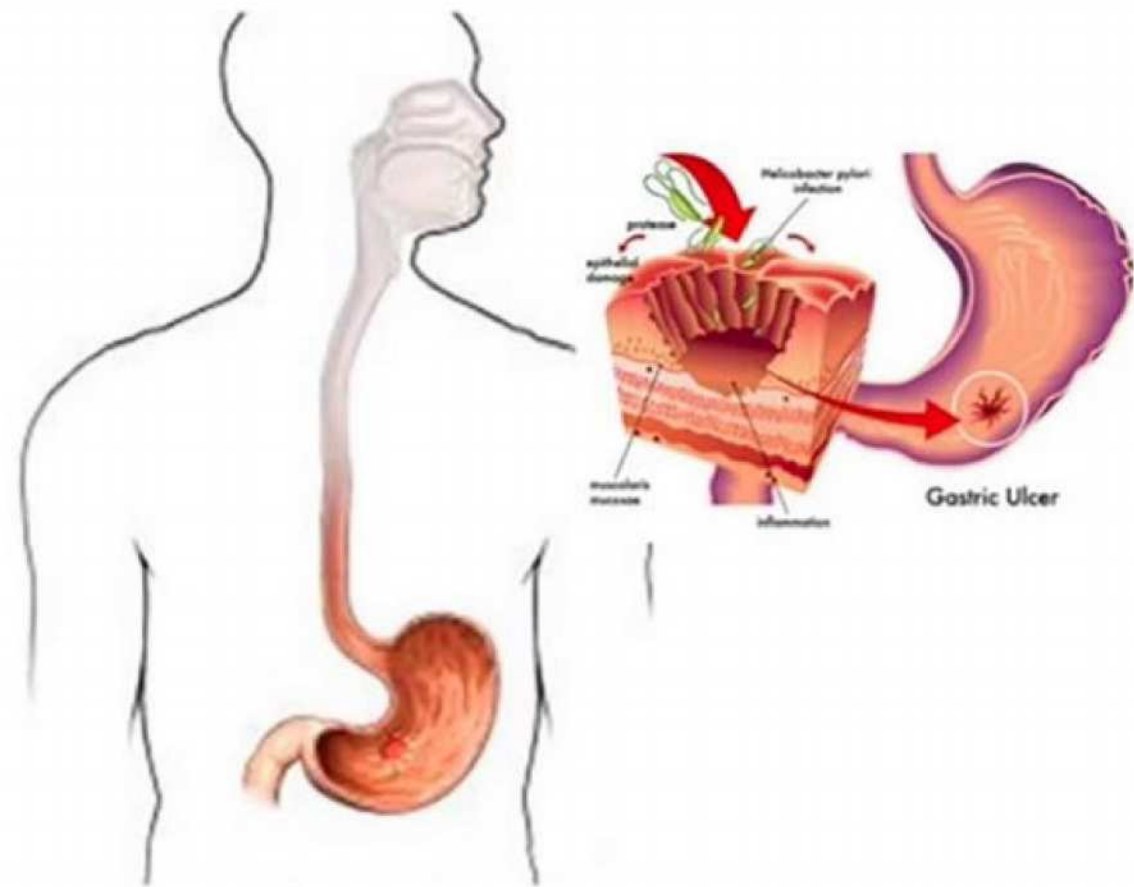
**Low serum
carotenes is a
good indicator
for MAS**

Treatment

- ① Diet: Decrease fat intake and Cellulose- Use MCTG
- ② Drugs:
Vitamin B12 + Folic acid in cases of BLS
Cholestyramine
Cortisone (small dose) may +++ enzymes
- ③ Replacement of Pancreatic Enzymes (if deficient)
- ④ Bile salt replacement (if deficient)
- ⑤ Specific treatment for the cause:
Gluten free diet Gluten Enteropathy
Antibiotics for Whipple Disease

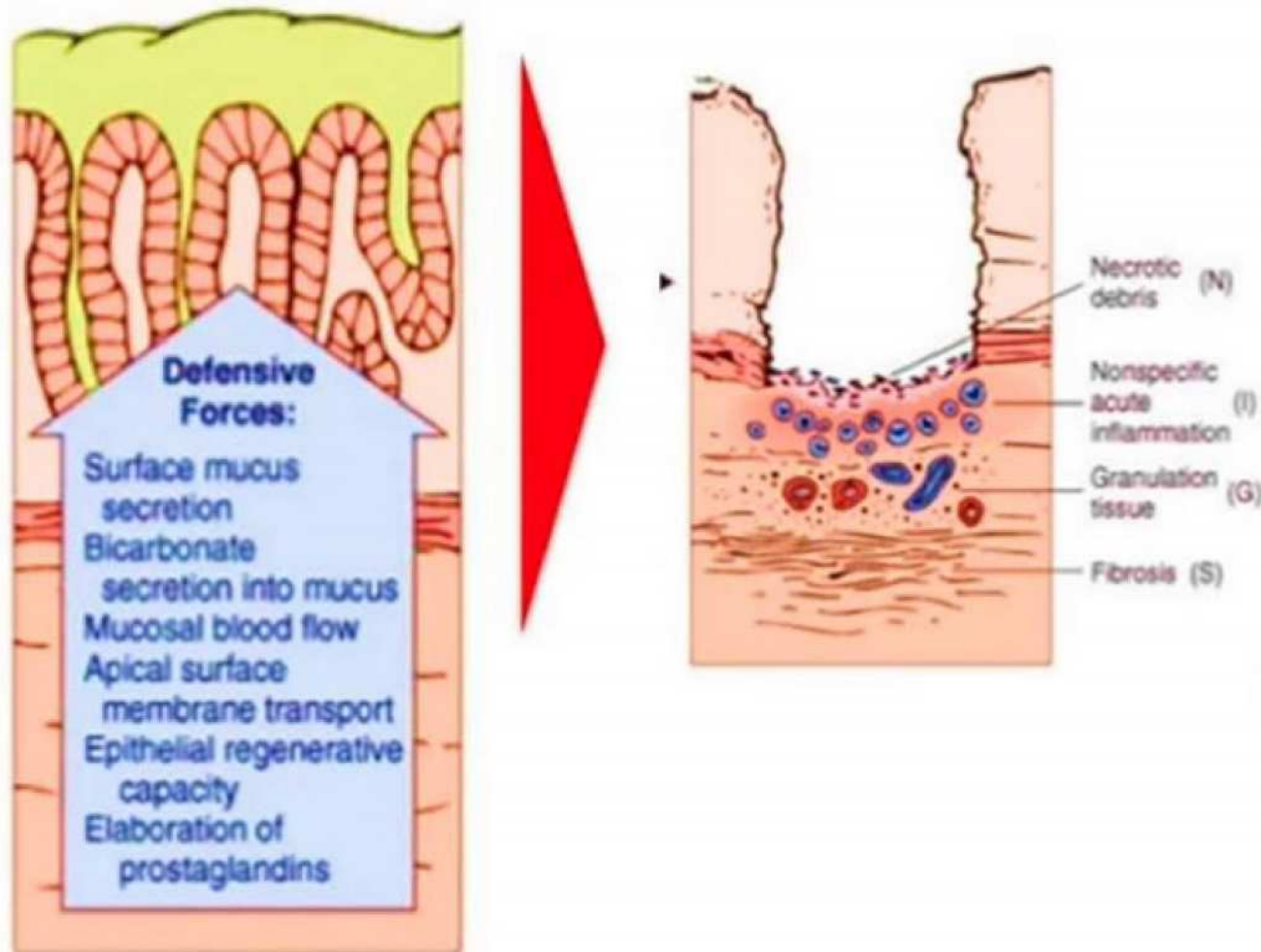


Peptic Ulcer



Definition

Peptic ulcer is a break in gastric or duodenal mucosa



Aetiology

The problem occurs when the normal mucosal defense factors (e.g. gastric mucosa) is impaired or overwhelmed by aggressive luminal factors such as increased HCL.

These factors include:

① **H. pylori infection:**

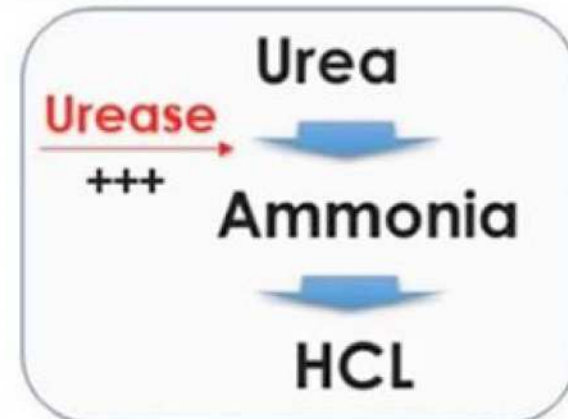
Increased urease production

Release of enzymes that destroy gastric mucosal barrier

Produce inflammatory mediators that aggravates the problem

② **NSAI**

③ **Gastrinoma**



Clinical Picture

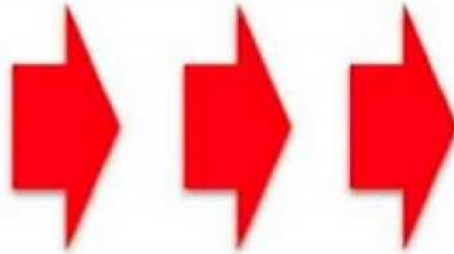
Heartburn

Pain

Hematemesis

Perforation

Stricture



Predisposing Factors

Cigarettes

Corticosteroids

COPD

Ca elevation

Cirrhosis of the liver [Alcohol]

Pain

- ① Localized
- ② Burning or hunger-like
- ③ Mid epigastric area slightly off center (Right)
- ④ Typically begins few hours after meal
- ⑤ Relieved by food or antacids

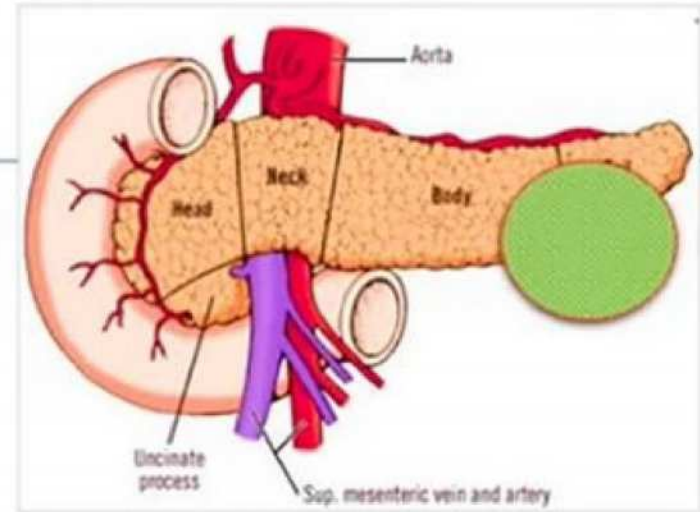
Notes

Note 1: if severe epigastric pain
+ Tenderness
+ Board like rigidity
...?! **Perforation**



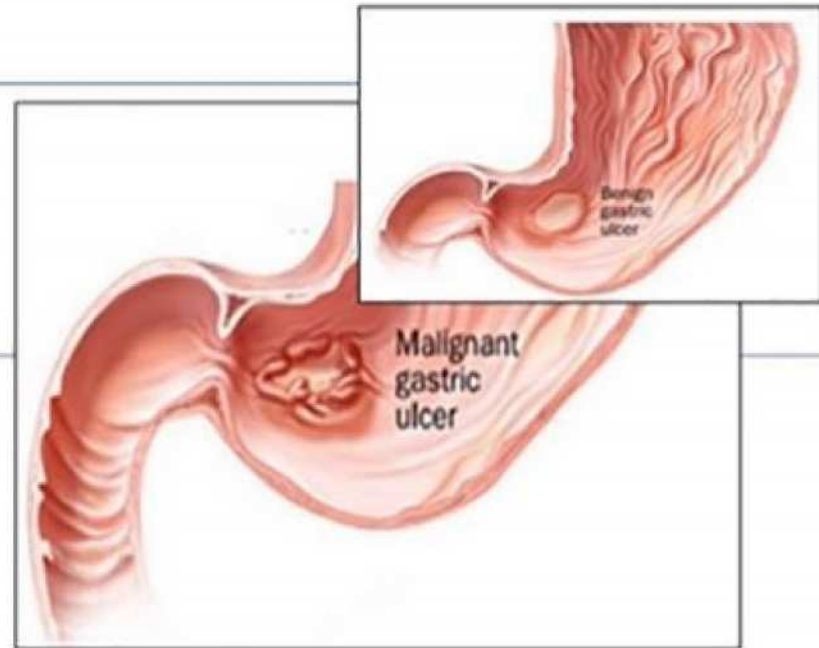
Notes

Note 2: if associated with Diarrhea
....?! Gastrinoma



Notes

Note 3: if associated with rapid weight loss....
?! Malignancy



Investigations

- 1 Endoscopy
- 2 Occult blood in stools
- 3 Detection of H.pylori by Elisa fecal antigens
- 4 C13 and C14 Urea breath test for H. Pylori

(Urease Test)



Investigations

- 1 Endoscopy
- 2 Occult blood in stools
- 3 Detection of H.pylori by Elisa fecal antigens
- 4 C13 and C14 Urea breath test for H. Pylori

(Urease Test)



Treatment

Rapid Relief of Pain

Anti Acids

Adjuncts to therapy
Al- containing antacids
can cause rapid relief of
symptoms



Treatment

Healing of the ulcer

- 1 Anti-secretory agents:
PPI -H2 Receptor antagonists

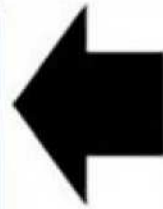
PPI: Inhibits H-K ATPase which blocks HCL production

H2 Receptor antagonists such as Cimetidine or Ranitidine

- 2 Eradication of H.pylori
Combination therapy of:
Antibiotics (Amoxicillin or Clarithromycin) + PPI + Bismuth

Treatment

Prevention of
NSAI induced
ulcers



Misoprostol

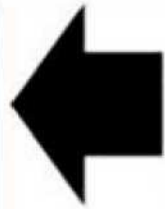
a **PG analogue** that stimulates gastrointestinal mucous production and enhances Bicarbonate gastric secretion (buffer HCL).



Note: In patients who have peptic ulcer and a need to use pain killers you can use **COX II Inhibitors** to decrease the pain.

Treatment

Prevention of
NSAI induced
ulcers



Misoprostol

a **PG analogue** that stimulates gastrointestinal mucous production and enhances Bicarbonate gastric secretion (buffer HCL).



Note: In patients who have peptic ulcer and a need to use pain killers you can use **COX II Inhibitors** to decrease the pain.

Treatment

Seconded line
treatment



Sucralfate + Bismuth

Sucralfate: acts by coating the mucosal surface

Bismuth: it adds the benefit that it has anti- bacterial action against H. pylori.